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Archives of Neurology and Psychiatry

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STUDIES OF CEREBRAL FUNCTION IN LEARNING

V. THE RETENTION OF MOTOR HABITS AFTER DESTRUCTION OF THE SO-CALLED MOTOR AREAS IN PRIMATES *

K. S. LASHLEY, Ph.D.

MINNEAPOLIS

Since the area was first described by Fritsch and Hitzig,[1] the function of the electrostimulable cortex of the cerebrum has been the subject of almost continuous controversy. The experiments were immediately called in question through criticisms of the technic by Dupuy,[2] Sanderson,[3] Carville and Duret,[4] and others, or by abstruse metaphysical deductions such as were advanced by Hermann[5] who objected to the motor area as violating the "unity of mind." The work of Ferrier,[6] Carville and Duret and Hitzig soon established the fact of the electrical excitability of limited areas of the cortex, but immediately a new question arose. Fritsch and Hitzig had considered the excitable zone as motor, if we may translate the expression, "entry of single psychic functions into material" by such a term. In this they were followed by Carville and Duret, who described the motor disturbances following lesions in the area as "paralysie de la motricité volontaire corticale." Ferrier also considered the area as motor. But most of the early work had been done with dogs, and the sensory disturbances which appear in this animal after ablation of the stimulable area were emphasized by Schiff,[7] Nothnagel,[8] Munk[9] and

* From the Department of Psychology of the University of Minnesota.

1. Fritsch, G., and Hitzig, E.: Ueber die elektrische Erregbarkeit des Grosshirns, Arch. f. Anat. u. Physiol. 1870, pp. 300-332.

2 Dupuy, E.: Experiment sur les fonctions moteur du cerveau, Compt. rend. soc. de biol., 1888, pp. 1025-1027.

3. Sanderson, J. B.: Note on the Excitation of the Surface of the Cerebral Hemispheres by Induced Currents, Proc. Roy. Soc. **22**:368-370, 1874.

4. Carville, C., and Duret, H.: Sur le fonctions des hémisphères cérébraux, Arch. d. Physiol. **7**:352-490, 1875.

5. Hermann, L.: Ueber elektrische Reizversuche an der Grosshirnrinde, Arch. f. d. ges. Physiol. **10**:77-88, 1875.

6. Ferrier, D.: The Functions of the Brain, London, 1876.

7. Schiff, M.: Untersuchungen über die motorischen Functionen des Grosshirns, Arch. f. exper. Path. u. Pharmocol. **3**:171-179, 1875.

8. Nothnagel, H.: Experimentelle Untersuchungen über die Functionen des Gehirns, Arch. f. path. Anat. u. Physiol. **57**:184-227, 1873.

9. Munk, H.: Ueber die Funktionen der Grosshirnrinde, Berlin, 1890.

Hitzig,[10] who interpreted the disorders of movement variously as due to the loss of muscular and cutaneous sensitivity or to the loss of kinesthetic images of the movements to be performed.

Following the suggestion of Tamburini, Luciani and Seppili[11] advanced the view that the motor areas contained somesthetic projection fibers as well as motor elements, and developed a theory of the sensorimotor function of the sigmoid gyrus and rolandic areas. In this they were followed by Horsley,[12] Mott,[13] Dana,[14] Rothmann[15] and many others.

De Barenne,[16] in particular, has demonstrated the existence of marked sensory disturbances in the cat after application of strychnin to an area which widely overlaps the stimulable area, and the general correctness of Luciani's view for the stimulable areas of lower mammals does not seem open to question, although there may be some doubt as to whether the motor functions of the cortex in these forms are comparable with those of primates.

Recent more critical work with primates, however, shows that in them a further specialization has occurred with the development of the fissure of Rolando as a line of demarcation between centripetal and centrifugal projection areas.[17] The work of Schäfer,[18] Mills,[19] Grün-

10. Hitzig, E.: Physiologische und klinische Untersuchungen über das Gehirn, Berlin, 1904.

11. Luciani, L., and Seppili, G.: Die Funktion-Localization auf der Grosshirnrinde, Deutsche Ausgabe, Leipzig, 1886.

12. Horsley, V.: On the Analysis of Voluntary Movement, 19th Century **29**:857-870, 1891.

13. Mott, F. W.: The Sensory-Motor Functions of the Central Convolutions of the Cerebral Cortex, J. Physiol. **15**:464-487, 1893-1894.

14. Dana, C. L.: A Study of the Functions of the Cortex of the Motor Area of the Brain, J. Nerv. & Ment. Dis. **21**:761-785, 1894.

15. Rothmann, M.: Ueber die elektrische Erregbarkeit der Zentralwindungen, Monatschr. f. Psychiat. u. Neurol. **32**:489-502, 1912.

16. Barenne, J. G. D. de: Sensory Localization in the Cerebral Cortex, Quart. J. Exper. Physiol. **9**:355-390, 1916.

17. The lack of any persistent paralysis in rodents, carnivora and ungulates after destruction of the stimulable areas and the occurrence of partial paralyses after destruction of parts of the corpus striatum in lower forms suggest that the differentiation of function of precentral and postcentral gyri in primates may be due rather to the acquisition of primative striate functions by the cerebral cortex than to a division of cortical functions which overlap in lower forms.

18. Schäfer, E. A.: On the Alleged Sensory Functions of the Motor Cortex Cerebri, J. Physiol. **23**:310-314, 1898.

19. Mills, C. K.: The Separate Localization in the Cortex and Sub-Cortex of the Cerebrum of the Representation of Movements and of Muscular and Cutaneous Sensibility, J. Nerv. & Ment. Dis. **38**:595-619, 1901.

baum and Sherrington,[20] C. and O. Vogt,[21] Lewandowsky and Simmons,[22] Hoppe,[23] Cushing,[24] Franz,[25] and Leyton and Sherrington,[26] all points to the conclusion that primary excitability is limited to the precentral gyrus and that sensory disturbances rarely result from lesions confined to this area.

These studies have defined the centrifugal function of the cells of the precentral gyrus and have confirmed the view that the area is motor rather than somesthetic, but in spite of the fairly general recent agreement as to the location of "motor" area, there is a wide diversity of opinion concerning the significance of the "motor" function. The area is somehow concerned with the production of movement, but how it acts and what kinds of movement it controls, are still debated points. Ferrier[6] emphasized the "automatic" character of movements after destruction of the motor areas and held that "All reactions not so (automatically) organized, and still dependent upon conscious discrimination and exercise of attentive volition are effectually and permanently annihilated." Munk[27] classified movements according to their somatic distribution and maintained that small unilateral adaptive movements (Einzelbewegungen) are permanently lost after destruction of the motor areas. His definition of these movements is by no means clear, but his accounts seem to imply that the movements had been learned, and he denies that a dog can learn to give his paw after destruction of both motor areas. Wagner[28] maintained that the chief function of the motor areas is in learning, and that animals deprived of them are incapable of forming any new habits. Bechterew[29] also refers to experiments lead-

20. Grünbaum, A., and Sherrington, C. S.: Observations on the Physiology of the Cerebral Cortex of the Anthropoid Apes, Proc. Roy. Soc. **72** B:152-155, 1903.

21. Vogt, C., and Vogt, O.: Zur Kenntnis der elektrisch erregbaren Hirnrindengebiete bei den Säugetieren, J. f. Psychol. u. Neurol. **8**:277-456, 1907.

22. Lewandowsky, M., and Simmons, A.: Zur Physiologie der vordern und hintern Zentralwindung, Arch. f. d. ges. Physiol. **129**:240-254, 1909.

23. Hoppe, H. H.: A Critical Study of the Sensory Functions of the Motor Zone (Pre-Rolandic Area): More Especially Stereognosis, J. Nerv. & Ment. Dis. **36**:513-527, 1909.

24. Cushing, H.: A Note Upon the Faradic Stimulation of the Postcentral Gyrus in Conscious Patients, Brain **32**:44-54, 1909.

25. Franz, S. I.: Variations in Distribution of the Motor Centers, Psychol. Monogr. **19**:80-162, 1915.

26. Leyton, A. S. F., and Sherrington, C. S.: Observations on the Excitable Cortex of the Chimpanzee, Orang-Outan and Gorilla, Quart. J. Exper. Physiol. **11**:135-222, 1917.

27. Munk, H.: Ueber die Fühlsphären der Grosshirnrinde, Sitzungsber. d. Berlin Akad. Wiss., 1892, 679-723; 1893, 759-781; 1894, 823-833; 1896, 1131-1159.

28. Wagner, V.: Discussion in Neurol. Zentralbl. **24**:1022, 1905.

29. Bechterew, W. v.: Die Funktionen der Nervencentra, Jena **3**, 1911.

ing to the same conclusion. He states that after removal of the motor centers associative motor reflexes are lost. "With unilateral destruction of the motor region in dogs the associative motor reflex may be elicited in the homolateral fore-leg, but in the contralateral fore-leg the reflexes learned earlier are forever lost and can not be reëstablished even after a number of conditioning associations (p. 1551ff.)." Gierlich [30] also supports this view of the exclusive motor function of the stimulable areas.

In opposition to these results, several writers have reported the acquisition of habits after the destruction of the motor areas or of the pyramidal tracts. Starlinger [31] trained a dog to give his paw after total destruction of both pyramidal tracts. Rothmann [32] observed learning in a rhesus monkey in which one precentral gyrus had been extirpated and the pyramidal tract of the other had been sectioned in the cervical region. Franz and Lashley [33] and Lashley [34] found learning ability in the rat unaltered by total destruction of the stimulable cortex. This result has been confirmed by Jellinek and Koppányi.[35]

In the contradiction of evidence here, we must favor the positive results. Failure to learn may be due to any one of a number of factors in addition to specific destruction of tissue, and a single positive case with certain destruction of the motor area is sufficient to discredit any number of negative findings such as are cited by Bechterew and Munk. It seems quite certain that the formation of conditioned motor reflexes is possible in the absence of the electrostimulable cortex, but this fact fails to reveal the normal function of the area in the performance of complex activities. Both Rothmann and Brown [36] seem to believe that in the intact animal the motor areas form the chief centrifugal path for complex adaptive reactions and that when learning occurs in their absence it is to be considered as due to vicarious function of other

30. Gierlich, N.: Ueber Symptomatologie, Wesen, und Therapie der hemiplegischen Lähmung, Wiesbaden, 1913.

31. Starlinger, J.: Die durchschneidung beider Pyramiden beim Hunde, Neurol. Zentralbl. **14**:390-394, 1895.

32. Rothmann, M.: Ueber die physiologische Wertung der cortico-spinalen (Pyramiden) Bahn, Arch. f. Anat. u. Physiol. (Physiol. Abt.) pp. 217-275, 1907.

33. Franz, S. I., and Lashley, K. S.: The Retention of Habits by the Rat After Destruction of the Frontal Portion of the Cerebrum, Psychobiol. **1**:3-18, 1917.

34. Lashley, K. S.: Studies of Cerebral Function in Learning, Psychobiol. **2**:55-135, 1920.

35. Jellinek, A., and Koppányi, T.: Lernfähigkeit gehirnverletzter Ratten, Anzeiger d. Akad. d. Wiss., Wien, 1923, No. 17.

36. Brown, T. G.: Studies, XXVII. 6. The Motor Activation of Parts of the Cerebral Cortex Other Than Those Included in the So-Called "Motor" Areas in Monkeys, Quart. J. Exper. Physiol. **10**:103-143, 1916.

parts rather than as an expression of their normal function. On the basis of Brown's work and of clinical evidence, however, Monakow [36a] is inclined to minimize the importance of the pyramidal areas for "voluntary" movement. He suggests that "We must give up or essentially modify the view that the precentral convolution alone conducts impulses to voluntary movement. It is probable that the pyramidal areas serve less for the execution of voluntary movements than for the inhibition of the kinetic functions of spinal coordination. Their function seems to be in the class of reflex activity." Lashley [37] has reported the survival of visuomotor habits after the complete destruction of the stimulable area and serious injury to the caudate and lenticular nuclei [38] and has suggested that the primary function of the stimulable area is the reflex regulation of postural and kinetic mechanisms. Similar results have since been obtained for the stimulable cortex with problem-box and maze habits.

Thus we find in the literature claims that the electrostimulable cortex is motor, that it is sensory, that it is sensorimotor, that its motor function is exercised through the storing of images of movement, that it is the final common path for all voluntary movements, and that it is a reflex center not primarily concerned with voluntary activity.

Much of the literature on the function of the electrostimulable cortex, as on cerebral localization in general, presents an inextricable tangle of physiologic fact and psychologic speculation. The long controversy between Goltz, Munk, and Hitzig was largely due to their inability to grasp each other's psychologic theories, and recent progress in psychology tends to invalidate much of the cerebral localization which was based on older conceptions of mental faculties.

Images have fallen into disrepute, and even the psychologists who still deal with them deny that kinesthetic imagery has any demonstrable relation to the initiation of movement (Thorndike [39]). The conception of volitional activity is too vague to have any scientific value. At best it represents an indefinite distinction between more or less complexly conditioned activities, and the conception of conditioned reflexes leaves the distinction without significance. The "will" has been largely discarded in psychology, although it is still in good standing in neurologic discussions. Such a statement as that the stimulable cortex is not

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36a. Monakow: See footnote 57.

37. Lashley, K. S.: Studies of Cerebral Function in Learning. The Motor Areas, Brain **44**:255-286, 1921.

38. When these experiments were reported, I believed that the lesions were largely confined to the caudate nuclei. Dr. J. B. Jonhston has since called my attention to the fact that the caudate nucleus in the rat comprises only a narrow median band in the corpus striatum and that the lesions reported actually included a considerable portion of the lenticular nucleus.

39. Thorndike, E. L.: The Mental Antecedents of Voluntary Movement, J Philos., Psychol. and Sc. Meth. **4**:40-42, 1907.

motor, but "psychomotor," means no more than that the pyramidal cells excite patterns of spinal motor cells rather than individual cells. It contributes nothing to our understanding of cerebral function. In the present state of psychologic science, we can not do better than follow the dictum of Bubnoff and Heidenhain,[40] "Es will uns überhaupt scheinen als müste die Untersuchung der physiologischen Processe in dem Gehirn von den jene Vorgänge begleitenden Bewusstseinsvorgängen möglichst absehen, wenn es sich um eine Deutung physischen Geschehens handelt," and rigidly exclude from neurologic discussion every subjective concept which cannot be translated into objective terms.

There is no evidence for the localization of any "mental function" in any part of the cerebrum. All that can be concluded from the existing evidence is that the conducting pathways concerned in particular kinds of behavior lead from receptor to effector through certain cerebral areas. Cerebral motor localization is a problem of the origin and function of the centrifugal neural impulses of the cortex. Their "volitional" or "automatic" character can be defined only in terms of their complexity of organization and their relative importance in the total motor integration or kinetic melody, and until so defined the terms are meaningless. The "reflex" conception of cerebral function, although still a theory and notably inadequate to account for all the phenomena of cerebral function because of oversimplification in its formulations,[41] is too well supported by evidence on nerve conduction and analogy with spinal functions to be disregarded in favor of any speculations concerning the localization of "psychic" functions.

STATEMENT OF PROBLEM

Stated objectively, three mutually incompatible theories concerning the function of the precentral gyrus are to be found in the current literature. They are: 1. This area is the only centrifugal outlet from the cerebral cortex for complexly integrated movements or for movements acquired as a result of training (voluntary movements or conditioned reflexes). 2. In the intact animal, the Betz cells are the principle centrifugal paths, but some neural impulses of like function may descend by extrapyramidal tracts, and these tracts may assume vicariously all the functions of the motor area. 3. The motor area is a part of the mechanism functioning in the regulation of tonus and posture and is not directly concerned in conditioned reflex activity.

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40. Bubnoff, N., and Heidenhain, R.: Ueber Erregungs- und Hemmungsvorgänge innerhalb der motorischen Hirncentren, Arch. f. d. ges. Physiol. **26**: 137-200, 1881.

41. The data on direct adaptation of unpracticed organs to the solution of problem-boxes presented later in this paper seem wholly inexplicable in terms of simple conditioned reflexes.

The first of these views is definitely ruled out by the evidence cited above for habit formation after destruction of the areas. The evidence for the second and third views, as applied to primates, is inconclusive, although a considerable mass of evidence derived from the cerebral paralyses seems to support the second. The validity of this evidence will be considered after the experimental data are presented.

A simple test of the hypotheses is possible. If after total destruction of the precentral areas an animal shows undiminished ability to carry out activities of all degrees of complexity, acquired before the operative destruction, this will be conclusive evidence that the lesion did not destroy any part of the conditioned reflex arcs involved in the activities, and, as a corollary, that these arcs do not traverse the motor areas. Loss of the conditioned reflexes following the lesion with their later reestablishment through training will support the second view, that the pyramidal cells of the precentral region are the principal efferent paths involved in habitual movements.

Such a test was made with the rat and gave unmistakable evidence against the participation of the motor areas in the activities of the simple maze and visual discrimination box (Franz and Lashley,[33] Lashley [37]). Since the publication of that report, the results have been confirmed for a complex maze and the "double-platform box." As the rat shows no paralysis after lesions to the stimulable areas alone, it is not possible to generalize from it to higher forms which do develop a paralysis. I have therefore repeated the tests in a series of experiments with monkeys.

EXPERIMENTAL METHODS

The parlysis which follows lesions to the precentral gyrus in monkeys necessitates a modification of the technic used with the rat. The general procedure was as follows: The animals were trained in rather simple manipulative acts involving a new and easily recognizable pattern and sequence of movements. They were then kept without practice for about two months, at the end of which time their retention of the habits was tested. This gave a measure of the normal loss to be expected from disuse of the habits over a period equal to that required for recovery from cerebral paralysis. After these *preliminary retention tests,* the motor areas were destroyed. The animals were then kept without further practice in the habits until the paralysis was so far improved that they were judged capable of making the movements required. They were then given a final series of retention tests (*post-operative retention tests*) and brought to necropsy.

Training Methods.—For training, the familiar problem-box method was used. The animals were confined in a large cage, 5 by 5 feet, to

the floor of which a small problem-box containing food was bolted. Three problem-boxes were used:

1. Pull box. The animal must reach through a circular hole, 2 inches in diameter, grasp and pull forward a rod which passed transversely 3 inches behind the hole. This released the lid of the box, which was thrown open by a spring.

2. Crank box. The animal must grasp and turn a crank projecting from the front of the box. The crank handle described a 6 inch circle. It offered a resistance of about 200 gm. at all points of the circle. It was set at "one o'clock" and must be turned counter-clockwise through 270 degrees, at which point it released the lid of the box. The lid was thrown back by a spring.

3. Hasp box. The animal must open an ordinary gate hasp, closed with a wooden plug inserted loosely through the staple, withdrawing the plug and lifting the hasp from the staple, over which it would fall again if released. He must then raise the lid of the box and hold it open while he reached in for the food.

Five trials a day were given with each box; the time spent in each trial was recorded, and detailed notes were made as to the use of right or left hand and the exact method employed in opening the boxes. Training was continued until the latches were released in a stereotyped manner without random movements. Several interruptions of training occurred lasting from one to several weeks, so that the learning curves do not represent the rate for continuous training.

In addition to the problem-box habits, each animal was trained to pick out cubes of banana from among cubes of wood of similar size and appearance. The cubes were scattered in irregular order under a sheet of glass supported 2 inches above the floor of the cage. They were placed about 6 inches back from the edge so that the animals had to reach under the glass to get the cubes, which they could see but could not distinguish by odor.

Operative Technic.—Destruction of the motor areas was made under ether anesthesia, with aseptic precautions. The region of the precentral gyrus was exposed by trephining and identified by electrical stimulation. The opening was enlarged by bone-forceps until the precentral gyrus and surrounding areas were exposed. Arm, leg and face areas were verified by stimulation, and the limits of the excitable area determined. The entire area was then undercut by thermocautery to a depth of about 6 mm. To avoid injury to the longitudinal sinus, a median bridge of bone, 1 cm. in width, was left intact. In one specimen the leg area of this region was undercut by passing the cautery diagonally mediad and downward until the resistance of the falx was felt, then cutting longitudinally across the gyrus. The dural flaps were then replaced and the wound closed. In the cases reported below the wounds healed without infection.

Retention Tests.—The animals all showed marked paralysis after operation. This cleared up gradually, and the retention tests were given about two months after the operation. (Numbers 1 and 3, v.i., recovered somewhat more quickly than is usually the case when the lesion is restricted to one hemisphere.) In the postoperative retention tests, the animals were placed singly in the large cage with each of the latch boxes in turn. The time required to open the boxes was noted, and the methods were recorded in detail for comparison with methods employed in learning and in the preliminary retention tests.

Verification of Lesions.—When the tests were completed, the operative fields were again exposed and explored by electrical stimulation. Excitable points found were mapped. The brains were then removed, fixed in 10 per cent. formaldehyd, and sketches made under a camera lucida. Serial sections of the region of the lesions were then prepared. Camera drawings of these were made and the lesions reconstructed from them.

PROTOCOLS

NUMBER 1.—This was a small male cebus, trained on the crank box, pull box and hasp box. The skull was trephined and opened on both sides in front of the precentral gyrus. The openings were extended backward to the fissure of Rolando. The leg, arm and face areas were located by electrical stimulation and destroyed by cautery. Cauterization extended beyond the stimulable area except in the median line. The wound was covered with mica and closed.

On the following day, there was a partial paralysis of both sides with great spasticity. Coordinated walking movements were possible, but there was great weakness of the legs. The arms were extended toward food. He could not grasp with his left hand. Partial grasping with the right hand appeared, but there was inability to raise food to the mouth. He recognized a banana, and made efforts to grasp it. The arms were usually hyperextended.

Four days later, he moved clumsily, his arms and legs spread out frequently, letting him fall prone. He grasped with his right hand, but was unable to hold food or lift it to his mouth. He ate by thrusting his mouth against the bread. He was well oriented in the room. Two weeks before operation he had learned to slip out of the crack as the door was opened and to run into an adjoining room. He did this twice on the fourth day. He had a tendency to stay near a cage containing other monkeys and to hide under it when pursued. There was marked tremor after slight effort.

Ten days after operation, he stood and walked without falling, fumbled in grasping, but was able to hold food in the right hand or to lift it to his mouth.

Thirty-five days after operation, he climbed and ran accurately, picked up small pieces of banana with the right hand without noticeable clumsiness, and made quick movements in efforts to catch flies. Retention of the problem-box habits was tested at this time.

The average time per trial in each day's practice (five trials daily) is given in Table 1 for each of the problem-boxes. This is followed by the average time per trial on each day of the preliminary retention tests, and similarly for the postoperative retention tests.

TABLE 1.—*Average Time in Seconds Per Trial Consumed in Opening Each Problem-Box in Each Day's Practice of Training, Preliminary Retention Tests and Postoperative Retention Tests*

Training Tests			Preliminary Retention Tests		
Pull Box	Crank Box	Hasp Box	Pull Box	Crank Box	Hasp Box
136.6	206.2	135.2	1.6	3.2	44.4
5.4	290.0	34.4	3.0	1.6	81.8
1.8	47.2	10.0	2.4	11.0	14.0
2.0	12.8	18.0	1.2	2.4	
1.6	4.6			5.6	
1.2	3.8			2.6	
2.4	10.8			2.7	
2.0	27.6				
2.2	2.6				
1.6	4.8		Postoperative Retention Tests		
	6.0				
	1.8		Pull Box	Crank Box	Hasp Box
	2.8		2.8	63.6	31.0
	1.6		2.2	7.4	57.6
	2.2			4.4	
	2.6			3.8	

Tests for visual discrimination were made. There was no error in 100 trials. At all times following the operation the animal was oriented in the cage and room and, with the exception of the motor disturbance, gave no indication of any deterioration.

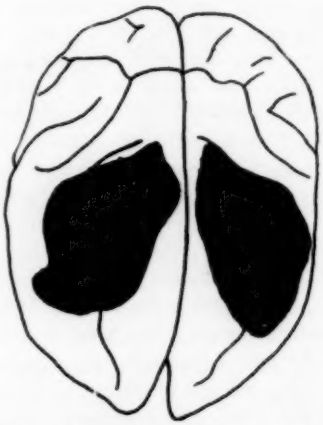

Fig. 1.—Extent of lesions in animal No. 1. Reconstructed from camera sketch and serial sections.

Extent of Lesions.—The areas destroyed are shown diagrammatically in Figure 1 and in sections in Plate 1.

Left Hemisphere: Mediad, the lesion began about 1 mm. behind the end of the fissure of Rolando and extended forward to the level of the knee of the corpus callosum. The cortex of the precentral gyrus was destroyed to within 2 mm. of the edge of the longitudinal fissure, but that of the median surface was uninjured. Caudad, the lesion extended slightly onto the postcentral gyrus but did not involve all of the cortex within the fissure of Rolando. Laterad, it extended to the upper border of the operculum. The parts of the stimulable area left intact were the paracentral gyrus, the cortex within the fissure of Rolando and the lateral part of the face area included on the operculum.

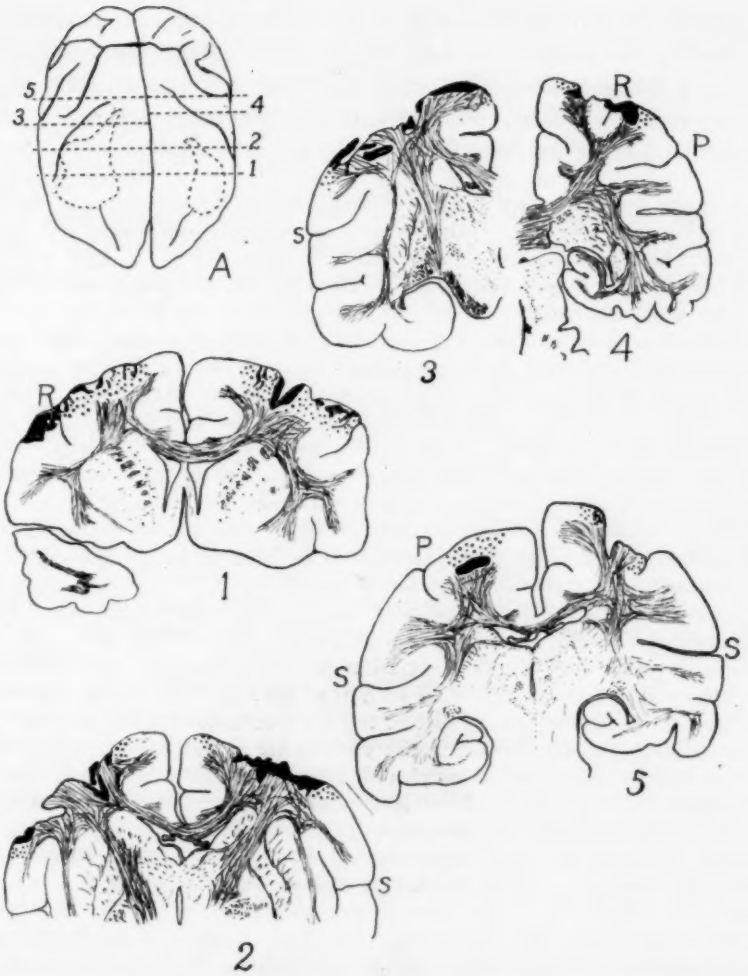

Plate 1.—Fig. *A:* Outline of the dorsal aspect of the cerebrum. The broken areas outlined indicate the extent of dural adhesions. The transverse broken lines show the level of the sections designated by the corresponding numbers.

Figs. 1 to 5.—Camera sketches of sections showing extent of lesions. Blood clots and scar tissue are indicated in solid black. Obviously degenerated cortex is marked with coarse stippling. *R*, fissure of Rolando; *S*, fissure of Sylvius; *P*, parieto-occipital fissure.

Right Hemisphere: The lesion was similar to that on the left but slightly more extensive. The cortex within the rolandic fissure was destroyed, and the lesion extended farther over the operculum.

After destruction of almost all of the arm areas of both sides and of most of the areas of the legs and face, this animal, on recovery from paralysis, showed perfect retention of visual and motor habits acquired before injury. Except for the paralysis and later spasticity, no significant change in his behavior could be noted.

NUMBER 2.—This was a small male cebus too wild for training at the beginning of the experiments. The motor area of the right hemisphere was exposed, the arm, face and leg areas identified and cauterized to a depth of 5 mm., caudad to central fissure, laterad and cephalad to a line 5 mm. beyond the limits of the excitable area.

Following operation, the left leg and arm were not used. The leg was hyperextended and gave some support to the body in standing or sitting, but made no stepping movements. No movements of the hand could be elicited. This complete paralysis of the left hand persisted for two weeks.

Four weeks after operation, the left leg was used almost normally. The left arm could be used to support the animal's weight, but tended to become rigid in hyerpextension, and the left hand could not be used for grasping.

Eleven weeks after operation, the paralysis had almost disappeared; the left hand was somewhat clumsy but could be used in grasping food. It was not used when the right hand was unrestrained.

Training on the problem boxes was begun at this stage of recovery. During the next three months, the problems were learned and retention tests given.

Seven months after the first operation, the left motor area was exposed and similarly explored and destroyed. Paralysis of the right arm and leg followed. It appeared to be as complete as that of the left side following the first lesion. It improved more rapidly, however. Six weeks after operation the right hand was used to pick up food, and, although still somewhat spastic, was judged capable of manipulating the latch boxes. Retention tests were therefore begun. The average time per trial for successive groups of five trials in training, preliminary retention tests, and postoperative retention tests are given in Table 2. Visual discrimination was unaffected by the operation.

TABLE 2.—*Average Time in Seconds Per Trial Consumed in Opening Each Problem-Box in Each Day's Practice in Training, Preliminary Retention Tests and Postoperative Retention Tests*

Training Tests			Preliminary Retention Tests		
Pull Box	Crank Box	Hasp Box	Pull Box	Crank Box	Hasp Box
24 hrs.*	126.0	102.2†	2.2	28.8	8.8
24 hrs.*	105.0	111.0†	2.0	1.6	6.4
2,520 sec.	16.0	235.0			
24 hrs.*	5.2	184.4	Postoperative Retention Tests		
366 sec.	7.8	19.6			
2.5	7.4	16.0	Pull Box	Crank Box	Hasp Box
1.4	1.4	55.6	9.8	14.8	102.5
4.5	1.2	6.0	3.6	11.4	93.4
20.4	2.8	6.6	1.2	7.2	35.4
1.6					60.6
					22.8
					15.4

* Failed to open box while under observation and was left in the cage over night.
† Time with hasp left unfastened.

The short time required for opening the boxes in the postoperative retention tests gives clear evidence for some retention of the habits. Much of the delay apparent was due to the weakness and clumsiness of the right hand. With each problem-box the attack in the postoperative retention tests was directly on the latches. The methods employed were at first those used before operation, and the movements were definitely adapted to solving the problems, although lacking force and accuracy; for example, efforts were all directed to turning the crank counter-clockwise.

The weakness of the right arm led to a surprising readjustment on the part of this animal. The operation on the right hemisphere made the left arm weak and spastic during training. In all of the trials of training and preliminary retention tests the left arm was used only as a prop, and the left hand was *not once* used in manipulating the latches of any of the boxes. After the second operation, the right hand was much more affected than the left, which had largely recovered, and an almost immediate shift to the left hand in opening all of the boxes occurred.

Pull Box: Postoperative retention tests. Trial 1: Fumbled in hole with right hand, removed hand, peered into hole; again inserted right hand and again pulled lever; twenty-seven seconds.

Trial 2: Right hand, fairly accurately, seven seconds.

Trial 3: He fumbled with right, then inserted left, grasped lever and pulled: seven seconds.

Trial 4: He inserted left hand at once; three seconds.

Trial 5: Left hand used at once; five seconds.

The right hand was used only four times in the succeeding forty trials.

Crank Box: Trial 1: He grasped the crank with his right hand at once and turned counter-clockwise. The crank stuck in the third quadrant. He pushed at it feebly, gave up, returned to the attack from the side of the box and pulled it through final segment; 160 seconds.

Trial 2: He grasped the crank with the right hand. Apparently, he was unable to move it. He grasped it with both hands and swung it around; twenty-five seconds.

Trials 3, 4 and 5: He used only the right hand and turned with difficulty moving to side of box and exerting direct pull instead of his former transverse rotary movement.

Trial 6: He grasped with the left hand and turned counter-clockwise; six seconds. All later trials were made with the left hand only.

Hasp Box: On the first three days of the postoperative tests, he pulled out the plug and disengaged the hasp promptly with his right hand, but lacked strength to lift the lid. He gave up after a few attempts with his right hand.

On the fourth day, he drew out the plug and disengaged the hasp with his right hand, then lifted the lid with his left foot. It fell back as he attempted to reach the food. He lifted it again with his left hand, climbed to the edge of the box, bringing his right side against the lid, so holding it up while he grasped the food with his left hand. On the second trial, he lifted with the left hand and held it up with his left hand, inserting his head for the food. In

all later trials, he lifted the lid with his left hand, sometimes holding it back with his left knee or with his head while reaching into the box with his left hand, or with the left hand while reaching with his head. On the eighth and all later trials, he pulled the plug with his left hand and used the right only as a prop.

Throughout the retention tests his activities were centered on the plug, hasp, and lid. He never attempted to lift the lid until the hasp was disengaged. When the lid was raised the next acts seemed definitely directed to holding it up, and though clumsy, the movements were clearly not random.

EFFECTS OF SUBSEQUENT DESTRUCTION OF THE CORPUS STRIATUM

A broad bladed cautery was next passed through the old lesion into the corpus striatum and drawn back and forth through this nucleus. The wound was closed, and the animal was kept under observation until his death eight days later.

On recovery from anesthesia, the animal showed marked spasticity of the left side. The left arm was usually hyperextended, although in walking or clinging to a perch the arm and leg assumed a normal posture. The left side was very weak, and when he walked the arm and leg frequently collapsed suddenly. He took food with his right hand and placed it in his mouth. When I held a bit of food, he drew my fingers to his mouth with the palm of his left hand, but without closing the fingers, which remained hyperextended.

On subsequent days, he used both right and left hands in walking, climbing and grasping food. The left side was spastic and very weak but capable of a variety of fine adaptive movements. The right side showed a coarse tremor, and athetoid movements of the left arm appeared when the right was used. There was marked paralysis of the pharynx. He kept his mouth stuffed with food or shavings but was unable to swallow.

At no time was the paralysis as marked as after the destruction of the cortex. Indeed the condition showed no resemblance to hemiplegia, but, except for the pharyngeal paralysis, was essentially that described by Wilson [42] for lesions of the striate nucleus without involvement of the pyramidal tracts. The possibility that the recovery from the initial paralysis was due to vicarious functioning of the striate nucleus seems thus definitely to be ruled out. The animal did not recover sufficiently for retention tests after this operation, but his behavior when released in the laboratory showed that his general orientation was unaffected.

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42. Wilson, S. A. K.: An Experimental Research Into the Anatomy and Physiology of the Corpus Striatum, Brain **36**:427-492, 1913.

When given an egg, he made efforts to break it by pounding it on the floor, as he had done before the operation, and in the performance of this habit both hands were used.

Extent of Lesions.—The extent of the destructions is indicated in Figure 2 and sections through the area are shown in Plate 2.

Right Hemisphere: The lesion extended cephalad from the median end of the central fissure to the middle of the superior frontal gyrus, bordering the longitudinal fissure but leaving the cortex of the median surface of the hemisphere intact. Caudad it invaded the postcentral gyrus and completely obliterated the fissure of Rolando. Laterad it extended well onto the operculum. Only the paracentral gyrus and the lateral portion of the face area remained intact.

The second operation destroyed all of the caudate nucleus and the greater part of the lenticular, leaving only the posterior end of the puramen intact.

Fig. 2.—The extent of the lesions in animal No. 2. Reconstructed from camera sketch and serial sections. The posterior border of the left precentral gyrus escaped injury.

Left Hemisphere: The lesion was less extensive than that on the right. It began 5 mm. in front of the median end of the central fissure and extended to the frontal lobe. All of the cortex of the median surface to the callosomarginal fissure was destroyed. The posterior edge of the precentral gyrus remained intact, for a width of about 5 mm.

In this animal, practically all of the precentral gyrus of the right hemisphere was destroyed. He was then trained in manipulative movements of the right hand. This was followed by partial destruction of the left precentral gyrus. On recovery from paralysis, he gave clear evidence of retention of the habits but owing to spasticity of the right hand, made a direct transfer of the habits to the left hand. The right precentral gyrus was almost completely destroyed, whereas a rather large proportion of the left precentral gyrus escaped injury, but in spite

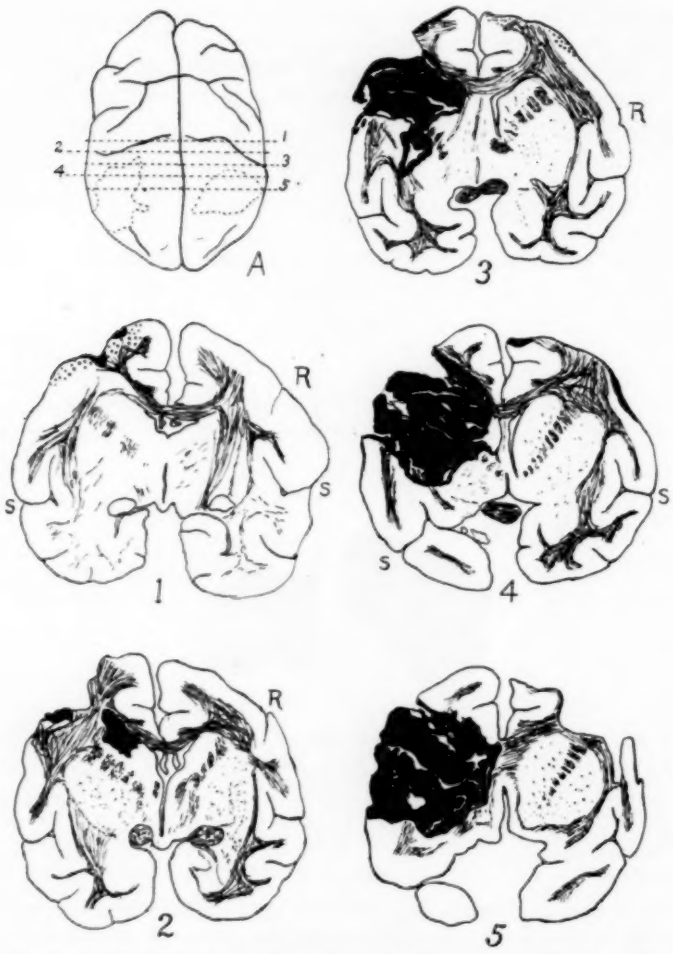

Plate 2.—Fig. *A:* Outline of the dorsal aspect of the cerebrum. The broken areas outlined indicate the extent of dural adhesions. The transverse broken lines show the level of the sections designated by the corresponding numbers.

Figs. 1 to 5.—Camera sketches of sections showing extent of lesions. Blood clots and scar tissue are indicated in solid black. Obviously degenerated cortex is marked with coarse stippling. *R*, fissure of Rolando; *S*, fissure of Sylvius; *P*, parieto-occipital fissure.

of this, the animal shifted to the use of his left hand in opening the problem-boxes. Subsequent destruction of the greater part of the corpus striatum did not produce a recurrence of the hemiplegic symptoms.

NUMBER 3.—This was a large female rhesus trained on problem-boxes and visual discrimination. After retention tests, the motor areas of both sides were exposed, mapped and cauterized. Following the operation the legs and left arm were completely paralyzed. The right arm made clumsy pawing movements. Twelve hours after operation, the animal walked a few feet with staggering gait, then collapsed with arms and legs widely extended, and for several days made no further efforts to walk. The following day she grasped a grape with her right hand and brought it to her mouth after several unsuccessful trials. The movements were clumsy and slow.

Four weeks after operation she seemed sufficiently recovered for retention tests, although still showing a general clumsiness and marked weakness of the left limbs.

The average time per trial for successive groups of five trials in training, preliminary retention tests and retention tests after operation is given in Table 3. Visual discrimination was unaffected by the operation.

TABLE 3.—*Average Time in Seconds Per Trial Required for Opening Each Problem-Box in Each Day's Practice in Training, Preliminary Retention Tests, and Postoperative Retention Tests*

Training Tests			Preliminary Retention Tests		
Pull Box	Crank Box	Hasp Box	Pull Box	Crank Box	Hasp Box
68.2	1,595.0	40.4*	1.0	4.2	24.0
2.6	111.8	373.2	1.0	3.8	6.6
1.0	145.4	124.6		1.6	5.0
1.0	304.2	24.2		1.2	3.6
	140.0	17.0		1.0	2.2
	16.8				2.8

Postoperative Retention Tests		
Pull Box	Crank Box	Hasp Box
1.0	2.2	49.2
1.0	1.4	9.8
2.6	1.4	8.4

* Time with hasp unfastened.

The time required to open the problem-boxes in the postoperative retention tests gives certain evidence of the retention of the habits. An average of 678.8 seconds was consumed in each of the first five trials of training in opening the boxes by the method of random activity. Only 17.5 seconds' average were required for the first five trials of the postoperative retention tests. The methods of opening the pull and crank boxes were the same before and after the operation. The persistent weakness of the left arm called for a change in method of opening the hasp box. Before operation, the animal had used the same method in twenty consecutive trials. The plug was pulled out of the staple with the right hand. The hasp was lifted from the staple with the right hand, turned back against the lid, and then transferred to the left hand. The lid was lifted with the left hand and the right hand

thrust into the box for the food. In the postoperative retention tests, the left hand was not used at all. She first lifted the lid with her right hand, then released it and attempted to grasp the food with the same hand, but the lid falling back prevented this. On the first trial, she finally held the lid back with her right hand, inserted her head in the box and took the food in her teeth. On the second trial, she thrust her head against the lid after raising it with the right hand and so held it open while the hand was inserted in the box. The same method was used on the third and fourth trials. On the fifth trial, she attempted to hold up the lid with her left foot and finally succeeded in this after overbalancing twice. In all later trials, she released the lid and allowed it to fall against her right arm as this was thrust into the box. These

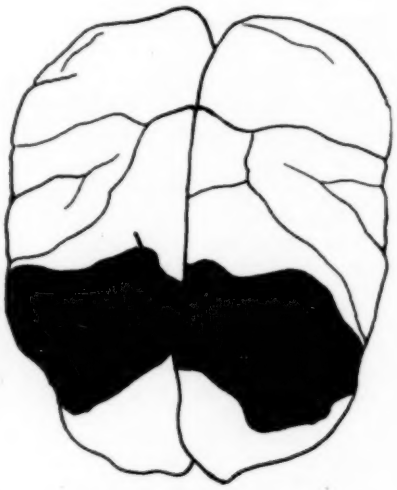

Fig. 3.—The extent of the lesions in animal No. 3. Reconstructed from camera sketch and serial sections. Practically the whole of both precentral gyri destroyed, including the paracentral gyrus and cortex within the central sulcus.

various acts were carried out with definite adaptation to the contour of the box and had none of the elements of random pulling and thrusting which characterize the initial stages of learning. As in the case of Number 2, there seemed to be an immediate adaptation of movements to opening the box, which had not been employed at any time in the previous practice.

Extent of Lesions.—The injured areas are shown in Figure 3 and sections in Plate 3.

Right Hemisphere: The lesion began at the posterior median end of the central fissure and included practically all of the precentral gyrus. On the median surface, all of the cortex above the callosomarginal fissure was destroyed. Practically all of the gyrus within the rolandic fissure was involved. Laterad.

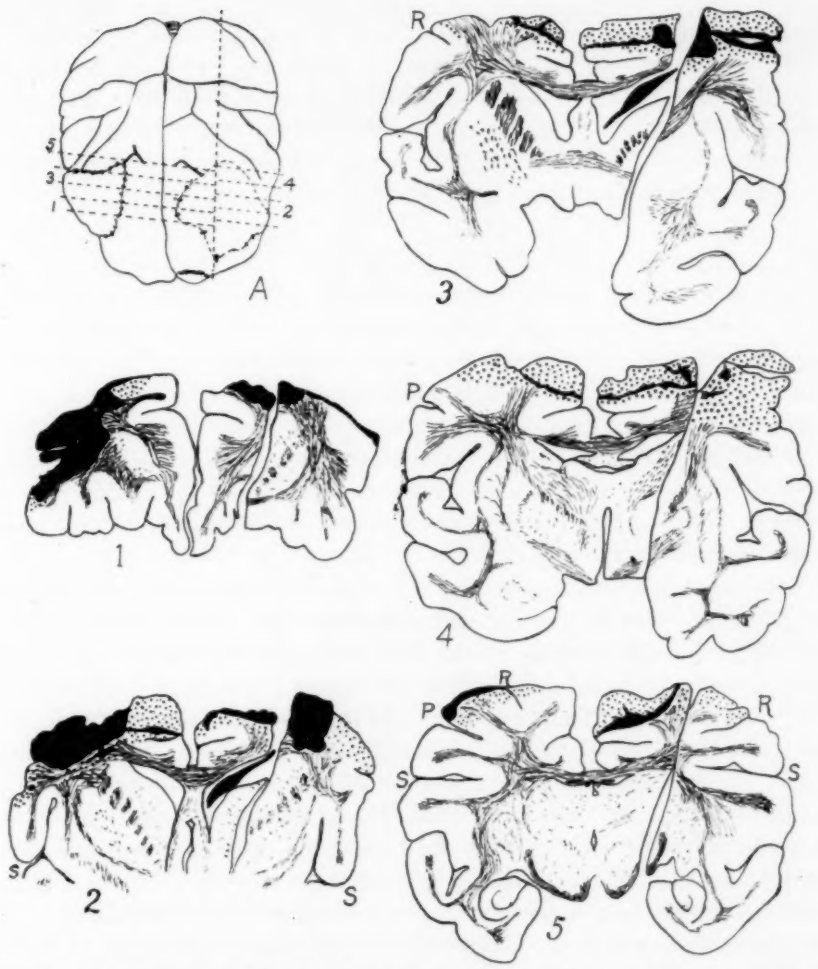

Plate 3.—Fig. *A:* Outline of the dorsal aspect of the cerebrum. The broken areas outlined indicate the extent of dural adhesions. In a preliminary examination, the left hemisphere was cut through along the longitudinal dotted line. The sections of its two halves are therefore from somewhat different levels. The transverse broken lines show the level of the sections designated by the corresponding numbers.

Figs. 1 to 5.—Camera sketches of sections showing extent of lesions. Blood clots and scar tissue are indicated in solid black. Obviously degenerated cortex is marked with coarse stippling. *R*, fissure of Rolando; *S*, fissure of Sylvius; *P*, parieto-occipital fissure.

the lesion included the upper half of the operculum. At most, only the lateral facial area escaped destruction.

Left Hemisphere: The lesion was almost coextensive with that on the right. More of the paracentral gyrus was destroyed and somewhat less of the operculum.

After practically complete destruction of both precentral gyri, this animal gave evidence of perfect retention of visual habits and habits of manipulation. Direct adaptive changes in behavior were made to compensate for weakness of the left arm.

DISCUSSION OF EXPERIMENTS

After extensive lesions to the precentral gyri of both hemispheres, each of the animals studied gave clear evidence of the retention of patterns of movement which had been acquired before the operative destructions. Evidence of this retention was obtained from a comparison of the time required for opening the problem boxes in initial training with that for the postoperative retention tests, from the restriction of reactions in the postoperative retention tests to the catches of the problem boxes, and from the persistence of individual peculiarities of opening the boxes.

The average time required by all animals for opening each box in the first five trials of training was 584 [43] seconds. An average of only 30.7 seconds was required in the first five trials of the postoperative tests. The animals all failed the hasp box in the preliminary training until first trained without the plug in the staple. In the postoperative tests, they all opened this box promptly with the hasp closed by the plug. At the beginning of the postoperative tests, each animal (except Number 3 with the hasp box) used the same hand or hands for each part of the manipulation as he had before the operation, and attacked the latches in his former manner, although the methods of attack were modified rapidly to compensate for the persistent motor difficulties. All the animals showed perfect retention in the visual discrimination test.

These results establish conclusively that the cerebral areas destroyed were not essential to the performance of the habits studied, and observations on the general behavior of the animals following recovery from the paralysis justify the further conclusion that the areas are not essential to the performance of any type of complex adaptive or habitual activity.

Four possible explanations of the results must be considered:

1. In no case did the operation destroy the entire precentral gyrus of both sides. The parts remaining intact may have contained a suffi-

43. This does not include the failures with the hasp box or the times when Number 2 was left in the cage over night.

cient number of fibers previously integrated in the habit to produce the conditioned reflexes, in spite of the great destruction of other fibers of equivalent function. Such a possibility is supported by data on other functional areas in which the various parts seem equipotential (Franz,[43a] Lashley [34]) and by the apparent equipotentiality of parts of the motor area revealed by electrical stimulation (Lashley [44]), but several facts speak strongly against this explanation.

Partial destructions usually entail a certain confusion in the performance of all the functions of an area, which seems to exceed anything of the sort noted in these animals.[45]

In Number 3, the destruction on both sides was so nearly complete that only a part of the face areas could have remained functional. If we attempt to explain the survival of habits as being due to the activity of undestroyed parts of the motor area, we must assume that a part of the face area is capable of performing all the functions of the entire motor cortex—an assumption which is as far from the accepted views of localization as is the denial of all habit function to the motor areas.

2. It might be urged that in the recovery from the motor paralysis, the vicarious functions assumed by other areas included the movements involved in the problem-box habits; that the habits were relearned during the period of recovery from paralysis. The habits, however, consist of particular patterns of movement associated with the stimuli presented by the latch boxes. During the postoperative period, there was no occasion for the animals to reacquire these particular patterns of movement and no opportunity for the movements to be associated with the latch boxes.

3. The long controversy concerning the sensorimotor function of both the precentral and postcentral gyri suggests that the two may both include centrifugal cells for the performance of habits. The literature cited in the first part of this paper seems to establish the differential function of the two areas, however, and the lack of paralysis after lesions to the postcenral gyrus makes the hypothesis untenable.

43a. Franz, S. I.: On the Functions of the Cerebrum: The Frontal Lobes, Arch. Psychol, 1907, No. 2, pp. 1-64.

44. Lashley, K. S.: Temporal Variation in the Function of the Gyrus Precentralis in Primates, Am. J. Physiol. **65**:585-602, 1923.

45. I am collecting data on this question at present. The evidence is not complete, but there is indication that, e. g., any extensive but incomplete destruction of the visual areas of both hemispheres in the rat is followed by inaccuracy of brightness discrimination, with great variability from day to day, such as has been reported by Franz in 1916 for aphasia, yet without any complete loss of any phase of the visual function. Such loss as appeared in the motor habits of the monkeys was almost certainly ascribable to the simple motor weakness, and gave no indication of an apraxia.

Nañagas [46] found a few islands of large pyramidal cells in the postcentral gyrus, but the great mass of them was restricted to the precentral. Finally, Brown [36] reported that the destruction of the postcentral gyrus did not abolish learning ability or interfere seriously with habits formed before the operation in the chimpanzee.

4. The only remaining possibility seems to be that the electrostimulable areas do not include the centrifugal elements of conditioned reflex arcs of any sort. (It is of course possible that they contain some such elements, but these cannot comprise any significant proportion of the total number of centrifugal cells, since their destruction leaves the habits completely unaffected.) In this, the experiments confirm for primates the results previously reported for the rat. The neural impulses involved in conditioned reactions do not pass from sensory projection areas to the precentral gyrus and thence to lower centers, but must be conducted by centrifugal cells lying outside of the pyramidal system. In the rat, the evidence points to the view that the centrifugal fibers of the sensory projection area itself are primarily involved in this motor function, since the destruction of any fourth of the cerebrum exclusive of the visual areas does not affect the performance of visual habits.[47] Whether or not the same lack of important transcortical conduction holds true for the monkey is questionable in view of the greater proportionate development of the transcortical association tracts in this animal, but it seems established that the "motor areas" are not concerned in the initiation of habitual movements.

THE CORPUS STRIATUM AND VICARIOUS FUNCTION

All recent students of the question agree that recovery from cerebral paralysis is not due to the assumption of the function of the destroyed motor cortex by the corresponding area of the opposite side. On the contrary, the simultaneous destruction of the areas in both hemispheres seems to be followed by a rather more rapid recovery than follows the destruction of either alone. The fact has been noted by Grünbaum and Sherrington [20] and by Wagner [28]. It was apparent in the slower recovery of Number 2 from the first operation than from the second. The

46. Nañagas, J. C.: Anatomical Studies on the Motor Cortex of Macacus rhesus, J. Comp. Neurol. **35**:67-96, 1922.

47. Experiments now in progress, which indicate that extensive fronto-parieto-temporal lesions may also abolish visual habits without producing a general deterioriation of learning ability, indicate that a mass action of the cerebrum is also somehow involved, but they do not seem to invalidate the conclusion that the efferent fibers of the sensory projection area are primarily concerned in the subcortical initiation of movements associated with the receptor for that area.

explanation is probably to be found in the forced practice which diplegia imposes on the paralyzed limbs (Odin and Franz [48]).

Other restricted cerebral areas have also been rather definitely excluded from participation in the vicarious function of the stimulable areas, by the work of Leyton and Sherrington [26] (Lashley [49]). Luciani [50] has suggested that recovery may be due to the activity of the corpus striatum, which has homologies with the stimulable cortex. This was tested in animal Number 2 of the present series by destruction of the right striatum after recovery from diplegia. Hemiplegic symptoms did not recur, so that we may conclude that the recovery had not been due to the vicarious activity of the striate nucleus.

THE FUNCTION OF THE ELECTROSTIMULABLE AREAS

The conclusions which may be drawn from these experiments are wholly negative. They seem to prove that the precentral gyrus does not include the efferent paths for learned activities; in the current localization terminology, it is not the center for "voluntary movements," as is almost universally assumed. But if this is true, what is the significance of the movements elicitated by electrical stimulation? How may we interpret the cerebral paralyses, and why do they especially affect the finer manipulative movements? A number of lines of evidence may help to answer these questions and clear up the function of the precentral gyri.

The Postural Function of the Stimulable Areas.—Many investigators have pointed out the similarity between the movements elicited by cortical stimulation and "voluntary movements." I am convinced that this is an error due to contrasting these movements with those which are elicited by stimulation of motor nerves or spinal cord. In the latter cases, the movements are wholly incoordinated, whereas the movements following cortical stimulation involve synergic groups of muscles. But in all cortical stimulation experiments which I have seen, the movements have been slow and rather massive, i. e., chiefly involving the larger musculature of the limbs. When smaller segments are moved, the movements are never coordinated as they are, for example, in grasping small objects. They never show the fineness of gradation and accuracy of adjustment which is characteristic of the movements of the intact animal. This has been observed by various investigators and interpreted as showing that the finer adjustments are integrated

48. Odin, R., and Franz, S. I: On Cerebral Motor Control: The Recovery from Experimentally Produced Hemiplegia, Psychobiol. **1**:33-50, 1917.

49. Lashley, K. S.: Studies of Cerebral Function in Learning. Vicarious Function After Destruction of the Visual areas, Am. J. Physiol. **59**:44-71, 1922.

50. Luciani, L.: Human Physiology, London **3**, 1915.

at some higher level and imposed through it on the motor area. But there is no direct evidence that this is the case. The movements following excitation are far more like the gross changes of posture which one may observe in the intact animal—the raising of an arm preparatory to snatching at food, bracing against a pressure, or the like. It seems significant that coordinated movements of the eyes are among the most easily elicited movements on electrical stimulation (although their stimulable points lie outside of the precentral areas), and that these movements in the intact man or animal are almost always a reflex fixation (postural adjustment) called out directly by exterostimulation and, in fact, can not be accurately performed in the absence of such stimulation, as with lids closed.

Wilson [51] has pointed out the similarity of the contractures in cerebral paralysis to the postural reflexes of decerebrate rigidity, and from this it seems certain that a part of the function of the stimulable areas is the regulation of these spinal and cerebellar postures. It seems rather probable that the movements obtained on electrical stimulation are only a further exhibition of this postural activity and are unrelated to the finer coordinations of conditioned motor reflexes, or motor habits.

The Dynamic Function of the Stimulable Areas.—The condition following lesions to the precentral gyrus or internal capsule, even in man, should be described rather as an enormous difficulty in making movements than as an absolute paralysis of movement. The degree of paralysis varies somewhat from day to day. Excitement seems to increase motor control (Minkowski,[52] Lashley [37]), and the paralysis may in part or wholly disappear during emotional disturbance, only to recur when the disturbing situation is past. If we may judge from the tonic condition of the muscles, there must be in excitement a general facilitation of lower motor centers which temporarily reinstates cerebral control. Further, if this is the case, in the intact animal in the absence of emotional stimulation cerebral control must likewise be conditioned by some such facilitation derived from the precentral gyrus. The work of Brown [53] and of Leyton and Sherrington [26] has shown that stimulation of the motor area does facilitate the centrifugal paths of other

51. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, Brain **43**:220-268, 1920.

52. Minkowski, M.: Etude physiologique des circonvolutions rolandique et parietal, Arch. Suisse de Neurol. et Psychiat. **1**:389-459, 1917.

53. Brown, T. G.: Studies in the Physiology of the Nervous System. XXV. On the Phenomenon of Facilitation. 4. Its Occurrence in the Subcortical Mechanism by the Action of Which Motor Effects Are Produced on Artificial Stimulation of the "Motor" Cortex, J. Physiol. **9**:131-145, 1915; also Footnote 36.

areas, either at cortical or subcortical levels, since stimulation of the precentral gyrus renders the otherwise inexcitable postcentral gyrus excitable for corresponding movements.

After partial recovery from cerebral paralysis, the most prominent symptom is the weakness of the formerly paralyzed limbs. The greater part of their repertoire of movements may be restored, speed may be nearly normal, but only a slight force can be exerted, and fatigue occurs readily. What is lacking in this condition is not an adequate integration of the motor impulses, but a sufficient mass of neural impulses to maintain muscular activity. This may be ascribed either to a reduction in the number of functional nerve cells, or to inadequate facilitation. The ready fatigability is evidence for the latter.[54]

There is evidence that the withdrawal of facilitation derived from other sources will produce similar weakness and fatigability and a tendency not to use the affected parts. Thus Munk [55] has shown that denervation of a limb has such effects, and Sherrington's [56] work has demonstrated that they are due to the withdrawal of impulses derived largely from the denervated muscles.

The importance of such facilitating systems has been emphasized by a number of recent investigators (Monakow,[57] Wilson,[42] Sherrington,[58] Tournay,[59] Hunt [60]). The general conception of these investigators is of a series of hierarchies of motor reflexes, all exerting a facilitating influence on the final common path. These involve at least the following elements.

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54. The all or nothing principle of nerve and muscle activity requires the assumption that strength of muscular contraction is dependent upon the number of motor fibers involved and the rate of succession of propagated disturbances. Piper's work (Elektrophysiologie menschlicher Muskeln, Berlin, 1912) indicates that fatigue involves a decrease in this rate rather than a reduction in the total number of muscle cells activated.

55. Munk, H.: Ueber die Folgen des Sensibilitätsverlustes der Extremität für deren Motilität, Sitzungsber. d. Berlin Akad. Wiss., pp. 1038-1077, 1903.

56. Sherrington, C. S.: The Integrative Action of the Nervous System, London, 1911.

57. Monakow, C. von: Aufbau und Lokalisation der Bewegungen beim Menschen, Ber. über d. iv. Kongress f. exp. Psychol. in Innsbruck, 1910.

58. Sherrington, C. S.: Postural Activity of Muscle and Nerve, Brain **38**: 191-234, 1915.

59. Tournay, A.: Conception actuelle des grande fonctions motrice, J. de Psychol. **17**:904-930, 1920.

60. Hunt, R.: The Static and Kinetic Systems of Motility, Arch. Neurol. & Psychiat. **4**:353, 1920.

1. Excitation of the motor cells supplying a muscle by impulses derived from the receptors in the muscle itself (Sherrington [56]).

2. Long spinal reflexes from synergic muscles (Sherrington,[56] Magnus [61]).

3. Other proprioceptive and general exteroceptive facilitation whose central mechanism is as yet rather obscure (Yerkes,[62] Richter [63]).

4. Vestibular and proprioceptive influences exerted through the mechanisms of the cerebellum.

5. Probably facilitation derived from thalamic mechanisms in emotional excitement (Head [64]).

6. Kinetic influences of obscure origin integrated in the corpus striatum (Wilson,[42] Hunt [60]).

Interference with any of these mechanisms is able to produce a change in the excitability of the final common path, and in the intact organism it seems certain that every act involves the participation of all of them, both by excitation and inhibition.

These considerations make it possible to form a tentative hypothesis concerning the function of the precentral gyrus. Its demonstrated facilitating effects, and its lack of direct participation in the conditioned reflex arc seem to throw it into a class with these other postural and tonic systems. Cerebral paralysis, is, I believe, to be interpreted as showing that a normal function of the stimulable cortex is to supply a substratum of facilitating impulses which act in some way to render the final common paths excitable by the more finely graduated impulses, descending from the cortex by extrapyramidal paths and producing the finer shades of adaptive movement. In other words, impulses descending from the precentral gyrus do not initiate the finer adaptive movements through the lower motor neurons, but only "prime" these cells so that they may be excited by impulses from other sources. The source of this activity and the probable interrelations of the stimulable areas with other parts of the motor system and with sensory projection areas present problems too complex for discussion here. Unquestion-

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61. Magnus, R., and de Kleijn, A.: Die Abhängigkeit des Tonus der Extremitätenmuskeln von der Kopfstellung, Arch. f. d. ges. Physiol. **145**:455-548, 1912. Magnus, R.: Welche Teile des Centralnervensystems müssen für das Zustandekommen der tonischen Hals- und Labyrinthreflexe auf die Körpermuskulatur vorhanden sein? Arch. f. d. ges. Physiol. **159**:224-250, 1914.

62. Yerkes, R. M.: Inhibition and Reinforcement of Reactions in the Frog, J. Comp. Neurol. & Psychol. **14**:124, 1904.

63. Richter, C. P.: A Behavioristic Study of the Activity of the Rat, Comp. Psychol. Monogr. **1**:1-55, 1922.

64. Head, H.: Studies in Neurology, London, 1920; Release of Functions in the Nervous System, Proc. Roy. Soc. **92** B:184-209, 1921.

ably, the areas receive excitations from other parts of the cerebrum [65] and it is probable that all parts of the kinetic system [66] are capable of mutual influence. Postural facilitation and inhibition may themselves be habitual responses, but the present experiments indicate that they are rather generalized and not independently organized for each specific manipulative habit.

Recent work in general tends to emphasize the complexity of neural functions. We must hesitate to ascribe an exclusive or precise function to any neural structure, for the evidence points rather to the view that observable behavior is always the product of the interaction of many neural systems and that the function of any system is dependent on its temporary physiologic relation to other systems. This is particularly true of the finer adaptive responses of the intact animal which are subject to inhibition and facilitation by innumerable factors. Their execution depends on preparatory postural adjustments, emotional and other dynamic facilitation, as well as integration of impulses from many exteroceptors.[67] The total mass of excitation is effective both through the specific efferent patterns activated and also through the general dynamic effects which alone are incapable of producing the overt motor reactions elicited. The experiments reported here indicate that the electrostimulable areas are rather more concerned with the maintenance of excitability and the regulation of postural reflexes than with the excitation and control of finely integrated adaptive movements.

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65. I have made several attempts to isolate the area from other parts of the cortex by circumsection but have not yet been successful. The literature on this point is conflicting. Marique (Brain **8**:536-538, 1885) reported the same results from circumsection as from excision of the area. Exner and Paneth (Arch. f. d. ges. Physiol. **44**:544-555, 1889) found similar results but were inclined to ascribe them to interference with the blood supply of the area. Schäfer (Jour. Physiol. **26**:23-25, 1901) reported one case of complete circumsection without paralysis. He does not report histologic examination of the lesion, however, and in view of the difficulty of the operation there is not sufficient evidence that the isolation was complete.

66. In this discussion, I have disregarded the important conception of static and kinetic functions advanced by Hunt (Arch. Neurol. & Psychiat. **4**:353, 1920) because the evidence does not show clearly to which of his systems the electrostimulable cortex is to be referred. The postural influences of the area would indicate a static function. What I have called the dynamic or "priming" function is rather a kinetic function, but is more primitive than the activities implied in Hunt's conception of the neokinetic system.

67. The statement that every act of the intact organism involves the participation of every neuron within the central nervous system is probably no more of an exaggeration than are the extreme theories of precise localization of function or of isolated conditioned reflex paths.

SUMMARY

The greater part of the precentral gyrus of both hemispheres was destroyed in monkeys which had been trained previously in habits of manipulation and visual discrimination. When the animals recovered from paralysis, it was found that they showed perfect retention of these habits. From this it is concluded that the so-called motor areas are not directly concerned with the performance of complex learned activities. The motor impulses of conditioned reflexes must descend from other areas of the cerebral cortex than the precentral gyri, and the latter cannot be regarded as the source of impulses to "voluntary movements."

Destruction of the corpus striatum subsequent to recovery from diplegia produced only the usual symptoms of striate lesion without recurrence of the symptoms of cerebral paralysis. Recovery from paralysis was therefore not due to vicarious function of this nucleus.

The evidence for considering the precentral gyrus as a part of the kinetic mechanism for reflex control of spinal posture and for maintenance of excitability of lower motor centers is summarized.

TUMOR OF THE HYPOPHYSIS*

REPORT OF A CASE

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AND
FRANCIS C. GRANT, M.D.
PHILADELPHIA

It is noteworthy that tumors of the hypophysis do not always cause symptoms that are purely hypophysial. In other words, regions of the brain may be involved that are more or less remote from the hypophysis; such, for instance, as the pons and cerebellum. This, of course, is to be explained by the varying size and position of such growths, and a confusing picture may result which causes difficulty in the diagnosis. But the converse may also be true; for an extensive hypophysial growth may invade rather remote regions and yet not cause such corresponding symptoms as might be expected. The case here reported is of the latter kind. The growth was very large and involved the interpeduncular space, the cerebellopontile angle, the pons and the cerebellum; yet the symptoms were mainly those that are now recognized as hypophysial. Such a case may lead to unexpected difficulties in the surgical operation.

An interesting question has also arisen in the present case as to the histology of the growth and its relation to the symptoms.

History.—A Greek, aged 24, was admitted to the Philadelphia General Hospital with weakness of the right arm and leg, and frequent headaches confined to the left side. The headaches began nine months before; they were paroxysmal and recurred several times a day. About three months after the onset he began to lose power in his right arm, and some months later still his leg became weak. The weakness progressed until he walked with some difficulty, his gait being slightly swaying. It was noteworthy that his hemiparesis came on gradually, accompanied with headaches.

The patient presented the appearance of the Fröhlich syndrome. He was over-fat, with a smooth face and feminine voice. He had shaved only once a month. He had been losing flesh (about 20 pounds [9 kg.]) before entering the hospital, at which time his weight was 170 pounds (77 kg.); so he must have weighed at one time almost 200 pounds (90 kg.). There was the so-called feminine rotundity and distribution of fat, and the breasts were enlarged and of the feminine type. The genitalia were normal, and the distribution of pubic hair was of the masculine type. This may be accounted for by the fact that his disorder did not appear until a good while after puberty. There were no skeletal changes, such as are seen in acromegaly.

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* Read at a meeting of the Philadelphia Neurological Society, Feb. 29, 1924. From the Neurological Clinic and Neuro-Pathological Laboratory of the Philadelphia General Hospital.

His personal history was unimportant, except for venereal infection, which was doubtfully syphilitic. The laboratory tests were negative, but the spinal fluid showed at times a heavy trace of globulin, and later there was xanthochromia.

The patient soon began to complain about his eyesight. Dr. Langdon, at his first examination, reported that the optic nerves were swollen and edematous;

Fig. 1.—Base of brain, showing the erosion of the pituitary fossa and the depth to which the tumor mass had infiltrated into the pituitary fossa and the adjacent sinuses. *A* is the area of erosion in the inner end of the petrous portion of the temporal bone where the tumor had passed over from the middle to the posterior fossa.

the veins were full, and there was a small hemorrhage in the left eye. Dr. Landgon continued to make frequent examinations; the papilledema was slowly progressive, but the most careful tests never showed a bitemporal hemianopsia. Later, however, there was a concentric contraction of the form fields. The pupils reacted slightly to light. Vision failed, but total blindness did not result.

The roentgen ray showed the sella to be extremely large, and the posterior clinoids were not demonstrable. There was increased tolerance for sugar. The basal metabolism rate was —21. The spinal fluid showed greatly increased pressure at times.

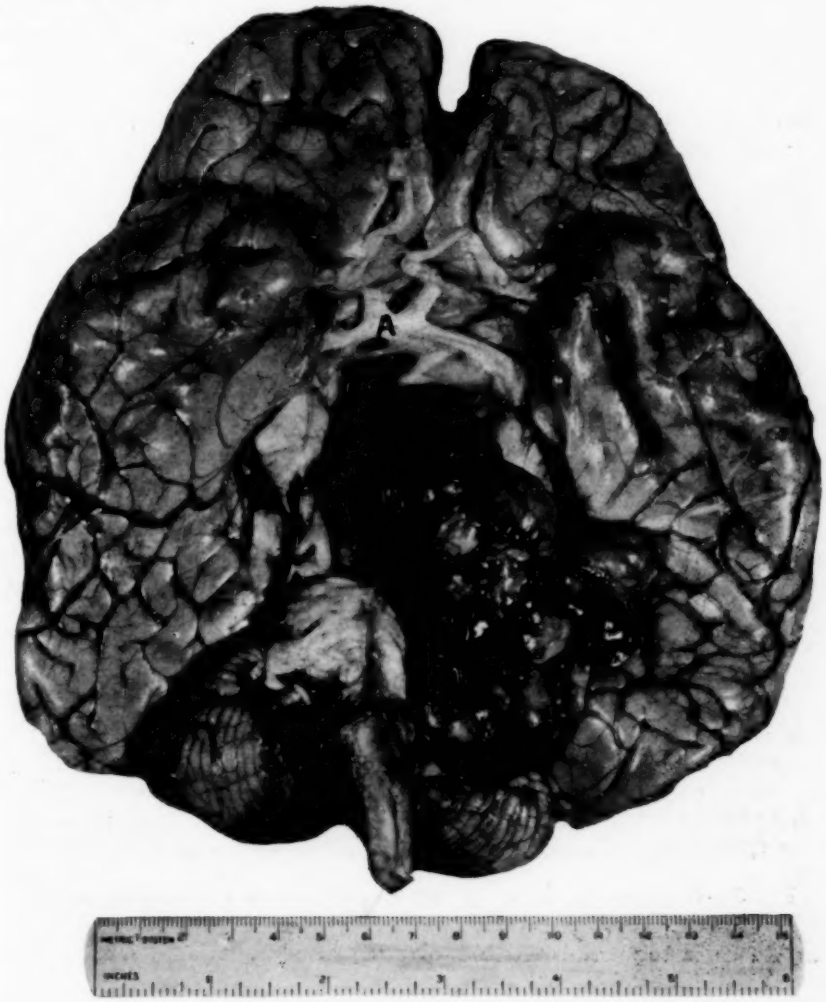

Fig. 2.—Base of brain with tumor in place, showing its origin and relation to adjacent structures. *A* indicates the optic chiasm much stretched but not definitely cut in two by tumor mass; *B*, position of free edge of tentorium which separated tumor into a supratentorial and infratentorial area.

This is a brief summary of the clinical history during the six weeks that the patient was under observation in the neurological wards. The diagnosis was a pituitary tumor; but at first, because of a possible history of syphilis, a short course of antisyphilitic treatment was tried, but without effect. The man's statement that he had had a venereal infection may have been a mere boast, as his potency was doubtful.

Operation was decided on, and was performed by Dr. Grant.

Operation (Dr. Grant).—The choice of the operative approach lay between a temporoparietal flap over the left motor cortex and a transfrontal approach in an attempt to reach the pituitary region by this route. The paralysis of the right side of the face and extremities on the right seemed to show an involvement of the left motor cortex. The enlarged pituitary fossa shown by the roentgen ray, the low basal metabolic rate, the sparsity of the beard, lack of axillary hair, the adiposity and loss of sexual power, all pointed to involvement of the pituitary gland. But the choked disks, and above all the concentrically contracted visual fields, seemed to prove that the optic nerves and chiasm

Fig. 3.—Tumor on removal. *A* indicates attachment of tentorium to tumor.

were only involved through general intracranial pressure, and not from localized pressure by a primary intrasellar tumor reaching up between the optic tracts. A subtentorial tumor, producing a bilateral symmetrical hydrocephalus, may cause sufficient dilatation of the third ventricle to erode the clinoid processes, press on the pituitary gland and produce marked symptoms of hypofunction of the gland. The tumor, if present, thus seemed to be located either in the left cerebrum or deep in the midline behind the pituitary fossa, either infratentorial or supratentorial. As compared to a transfrontal approach, a temporal flap exposing the left motor cortex was much the easier of the two procedures; if the tumor was situated here, removal would be possible, and by an intradural approach with elevation of the tip of the temporal lobe it was thought that we might reach the pituitary fossa from the side sufficiently closely to determine the presence or absence of a large growth.

Accordingly, a temporal flap to expose the motor cortex on the left side was thrown back. The anterior limb of this flap was placed within the hair line so that it might serve as the posterior limb of a transfrontal approach if this seemed indicated later. The dura seemed to be tense. Ventricular tap into the anterior horn relieved the intracranial tension entirely, and showed that the ventricle was very large. A complete exploration of the left cerebral cortex from the olfactory nerves anteriorly to the lateral sinus posteriorly was made. The hemisphere was lifted up until the base of the pituitary fossa was seen. Nothing suggestive of the presence of a tumor appeared. In fact, the extreme dilatation of the left lateral ventricle made it unlikely that the tumor lay within the cerebral hemisphere. Decompression was therefore performed at the base of the flap, and the wound was closed.

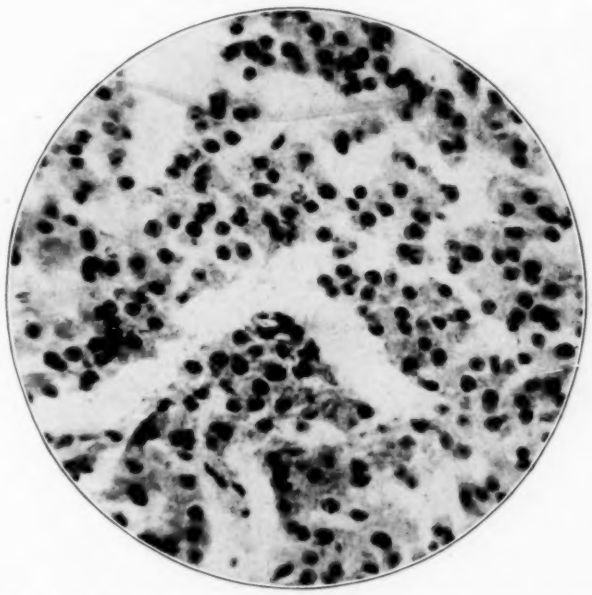

Fig. 4.—Stained section of tumor, showing its cellular structure.

We believed that the tumor lay principally in the midline. From the operative point of view, it was essential to discover whether it lay above or below the tentorium. Convalescence following the craniotomy was uneventful, except that the intracranial pressure had not been relieved to any extent by the large decompression at the base of the flap.

A ventriculogram was decided on as a means of locating the position of the tumor in its relationship to the tentorium. About 120 c.c. of fluid were removed and replaced by air. The marked symmetrical dilatation of both lateral ventricles may be seen in the illustrations. It is significant that the third ventricle was nowhere visible, the fourth ventricle and the basal cistern contained no air, and there was no air in the subarachnoid space. The diagnosis of tumor occluding the third ventricle was then made. Approaching this region was a formidable procedure requiring a large lateral flap, involving the tying off of all communicating veins between the cortex and the longitudinal sinus over one hemisphere, outward retraction of the hemisphere, incision through the

corpus callosum and ligation of the veins of Galen. As the constant intracranial pressure had raised up the bone flap and caused a pressure necrosis with a mild infection at the anterior angle of the wound, further surgical procedure was out of the question. The patient survived the ventriculogram only a week. His condition had been becoming steadily worse, so that the air injection cannot be entirely blamed for his death.

The tumor as revealed at necropsy was inoperable from its size and the degree of infiltration into the nasopharynx. The damage done to the left cerebral peduncle explained the left motor cortex symptoms. The retrochiasmal position of the tumor accounts for the lack of visual field defect, while the blockage of the third ventricle demonstrated the cause of the choked disks

Fig. 5.—Lateral view of ventriculogram plate, showing symmetrical dilatation of lateral ventricles, obliteration of third and fourth ventricles and absence of air in subarachnoid space: *A* indicates the inferior horn; *B*, the anterior horn; *C*, the area in which the third ventricle should have appeared on the plate if it had been distended with air; *D*, the posterior horn of the lateral ventricle.

and bilateral hydrocephalus. It is interesting to note that in spite of the extension into the left cerebellopontile angle, auditory and cerebellar symptoms were entirely absent. The tumor is interesting from its large size and the length of time it must have been present to attain such proportions while producing relatively few symptoms. If its position had been accurately localized before the growth had spread over into the left cerebellopontile angle and down into the nasopharynx, it might have been removed. The ventriculogram made the localization accurately. But, unfortunately, as is so often the case, tumors localizable in this way have reached such large dimensions that operative removal is out of the question. We present this case to show the many confusing elements which made the localization difficult, and how the tumor, in

spite of the fact that it apparently completely filled up the left cerebellopontile angle, gave no symptoms referable to this area, in which the presence of a tumor is usually easily determined.

Pathologic Report (Dr. R. B. Richardson).—The brain was slightly larger than normal. It weighed 1,430 gm. Some of this weight was due to the general edema, which was quite apparent. In consistency, the brain was soft and friable.

A tumor was seen in the region of the sella, impinging on the interpeduncular space. It extended over the posterior border to the left cerebellopontile angle. The mass was in two portions. One appeared rather spherical, with a smooth surface, and measured 4.5 cm. in diameter, and it had eroded deeply into the

Fig. 6.—Anteroposterior view of ventricular system after air injection, showing: *A*, symmetrical dilatation of lateral ventricles; *B*, obliteration of third ventricle. No air is seen in this region.

portion of the skull about the sella turcica. The other was an irregular, loosely knit mass, convoluted and triangular in shape from the pressure of the cerebrum above and the cerebellum below; it was attached to the spherical mass by a neck, which was due to the constriction of the tentorium. The posterior mass was about 1.5 times the size of the former. When it was raised from its bed in the cerebellum, it was seen to have indented by pressure the anterior portion of the left cerebellar lobe down to the peduncle, and to have left a deep imprint in the left lateral surface of the pons and left cerebral peduncle. The undersurface of the left temporal lobe, against which it lay, also showed depression and evidence of injury. The third and fifth nerves were intact. The chiasm had been slightly pressed on.

The left cerebral cortex showed three areas of softening, filled with a choroid plexus-like material. They measured 0.5 cm. in diameter. There was extensive erosion of bone. The sphenoidal and ethmoid spaces were destroyed, and a probe even extended under the skin. The lower part of the tumor mass must have pressed on the upper part of the pharynx.

The tumor sprang from the pituitary body and caused extensive erosion in the sella turcica area, expanding over the edge of bone posteriorly, eroding the clinoids, and filling the left cerebellopontile angle, with resulting dissolution of the anterior portion of left cerebellum, and pressure on the cerebral peduncle and pons of the same side.

Dr. N. W. Winkleman, of the Neuropathological Laboratory, reported: "Microscopically, the tumor is an adenoma. With differential staining methods, particularly Mallory's connective tissue stain, the tissue is found to stain very poorly, most of the cells taking the acid stain, though feebly. The tumor is, therefore, a chromophile adenoma of the acidophile type."

COMMENT

The tumor was of extraordinary size, the largest hypophysial tumor that we have ever seen—about 9 cm. long by 5 cm. in diameter. The microscope showed it to be an adenoma. The parts involved by it were not only the sellar and infundibular region, but the interpeduncular space, the pons, cerebellum, cerebellopontile angle, temporal lobe and the bones of the base of the skull. In spite of this wide extent, it presented a rather limited symptomatology.

There was an early right hemiparesis, which can be explained by the great size of the tumor, which caused it to press on the left cerebral peduncle. There must have been internal hydrocephalus also, caused by a complete blocking of the third ventricle and indicated also by the xanthochromia.

The Bárány tests, made by Dr. Hunter, seemed to indicate an involvement of the vestibular paths, and this can be explained by the fact that the tumor caused pressure on both the pons and the cerebellum.

It is rather remarkable that there was no bitemporal hemianopsia, but only a concentric narrowing of the form fields, which appeared later. It is strange that the growth did not cause complete blindness early in the case, but it lay somewhat behind the chiasm. The statement is made by some writers that papillitis is rare in pituitary tumor; nevertheless, a high grade of choked disk was observed in the present case.

Symptoms which were conspicuous by their absence were such as would have pointed to the interpeduncular space, the cerebellopontile angle, the pons, cerebellum and left temporal lobe. All these parts were invaded, yet there was no paralysis of the third, fifth, sixth or seventh nerve. There were no marked cerebellar symptoms, unless it was a

slight swaying walk, easily confused with the hemiparesis; nor were there any aphasic symptoms, such as might have been caused by a lesion of the left temporal lobe. The extensive necrosis of bone, without more severe pain, is noteworthy. Altogether, the case is remarkable as showing the extensive involvement that may be caused by a hypophysial tumor without a corresponding complication of symptoms. The Fröhlich syndrome was the most reliable sign and led to the correct diagnosis.

The value of the calorimeter in distinguishing between hyperpituitarism and hypopituitarism is claimed to be great. In acromegaly, according to Cushing, the basal metabolism may be as high as + 30, whereas in cases of insufficiency it may fall as low as — 39. In the present case, it was — 21.

It would have been interesting to examine the thymus and other glands of internal secretion, but permission to open the body, except to remove the brain, was refused. The pineal body was not enlarged.

Camus and Roussy, experimenting on dogs, have been led to deny most of the conclusions heretofore held by clinical neurologists.[1] The symptoms of hypopituitarism are caused, according to them, not by injury to the hypophysis, but by injury to other structures at the base of the brain, especially the infundibulum and tuber cinereum. They seem to think that the hypophysis has nothing to do, for instance, with polyuria. All this serves to show that opinions are still shifting on the subject of the hypophysis and its functions. But these laboratory results are to be accepted with caution.

Cushing[2] classifies tumors of the hypophysis as follows: chromophobe adenoma with hypopituitarism, chromophile adenoma with acromegaly, cystic adenoma with hypopituitarism, adenocarcinoma with hypopituitarism. He says that the question as to whether chromophobe adenoma is a real tumor or a functional hyperplasia has not been settled yet. In the present case, the laboratory report was to the effect that the tumor was of the chromophile variety. Nevertheless, the tissue stained poorly. Clinically, the case was not one of acromegaly, but of the Fröhlich syndrome. The question may be raised, in view of the conflicting opinions now being advanced about the hypophysis, whether it is not really the anterior lobe that is responsible for all these changes, whether of acromegaly or of Fröhlich's syndrome. It is this anterior lobe alone that is composed of glandular tissue, and thus allies the hypophysis with the other endocrine glands. The posterior lobe is composed of nervous tissue and may be largely vestigial.

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1. Camus, J., and Roussy, G.: Rev. Neurol. **38**:622 (June) 1922.
2. Cushing: Abstr., Pract. Med. Series **8**:217, 1922.

DISCUSSION

DR. EDWARD B. KRUMBHAAR: Although the hypophysis is anatomically divided into three parts (anterior, intermediate and posterior), for clinical purposes only two are considered; the anterior and the posterior. This lumping together of intermediate and posterior is a confession of ignorance as to which, if either, is responsible for such conditions as Fröhlich's syndrome. Assuming for the moment that the basal part of the brain is not specifically concerned with the clinical conditions under consideration in spite of the recent work (and the majority of evidence is still on the side of the hypophysis), it is possible to consider five syndromes: (1) hyperprepituitarism, (2) hypoprepituitarism, (3) hyperpostpituitarism, (4) hypopostpituitarism and (5) dyspituitarism.

The arguments in favor of such a classification have already been presented elsewhere.[3]

Group 1 is caused by either hyperplasia or adenoma of the acidophil type of chromophil cell, and Group 2 by its diminution. Basophil cell increase is rare, and its significance is not known. The chromophobe cell, aside from its increase in pregnancy, is not associated with specific changes, although a chromophobe adenoma, like a cystic adenoma, adenocarcinoma or any other tumor in that locality can cause "hypopostpituitarism" (Group 4) by pressure atrophy of the posterior lobe.

In the present case, therefore, the hypopituitarism can be explained in the same way. The absence of skeletal changes in the presence of an acidophil adenoma is not in accord with the theory, but is possibly explainable by the observation that the acidophil cells do not take the acid stain normally.

DR. W. B. CADWALADER: I have seen one tumor, growing from the pituitary, that I think was as large as the one in the present case; it occurred in a patient of Dr. Burr's, in a case of acromegaly shown before this Society a number of years ago. The absence of hemianopsia might be explained as suggested by Dr. de Schweinitz, who has shown that there are frequent variations in the relations of the pituitary to the chiasm.

DR. J. H. W. RHEIN: Some years ago I reported a case of tumor involving the pituitary body, in which the tumor was certainly as large, if not larger, than the one just exhibited. The tumor measured 2 by 2 by 2½ inches (5 by 5 by 6.9 c.c.), and had displaced the brain tissue upward so that the anterior horns of the lateral ventricle were pushed up as high as the knee of the corpus callosum. The sella turcica was necrosed, and the optic chiasm was stretched over the tumor. A curious feature in the case was the absence of symptoms indicating pressure, except atrophy of the optic nerve, although the peduncles were much flattened. Microscopic study of the tumor showed it to be an adenocarcinoma.

DR. TEMPLE FAY: I recall three cases from the service of Dr. Frazier studied during the past two years, which manifested symptoms somewhat similar to those shown in this case. In all, operation was performed over the parietal lobes without disclosing a growth. The symptoms were those of parietal lobe lesion: numbness and tingling in the hands, astereognosis, loss of the sense of position, and dysmetria with some adiadokokinesis. In one case, the patient complained of numbness and tingling over the entire half of the body. Subsequent exploration or necropsy revealed in all three a tumor behind the chiasm and between the peduncles. The symptoms which had misled us in localization were probably due to pressure on the sensory tracts of the peduncles by the

3. Krumbhaar, E. B.: Med. Clin. of N. Am., December, 1921 (also Arch. of Diagnosis, April, 1922).

tumor, or by pressure on the end surface of the optic thalamus. Our records show that symptoms such as this man gave of hemiparesis, or the sensory symptoms, shown in our three cases, are rare in pituitary tumors, and hence this seems to be the most interesting and unusual feature in the present case.

Dr. Charles K. Mills: In the light of this case and three cases referred to by Dr. Fay, and three cases recently reported, in all of which operation guided by focal diagnosis failed, it appears that we have not come to the end of that old subject of cerebral localization. Even the manner of naming these cases may be misleading. The cases reported were called "suprasellar," and a too intense focusing on the pituitary has led to mistake. We need a new symptomatology of lesions of the lateral ventricle. The operations performed in most of these cases were for a parietal lobe neoplasm. A negative discussion of this kind may not lead anywhere immediately, but it should result in a better localization of tumors in the third ventricle.

Dr. J. Hendrie Lloyd: The important point in diagnosis in this case is the localization. I think you will observe that the great extent of this tumor tends to make the diagnosis confusing. In the first place, the disorder began with hemiparesis, which is not an ordinary early symptom of pituitary tumor; but it is explained by the pressure on the left cerebral peduncle. The growth extended backward into the interpeduncular space, and also made pressure on the cerebellopontile angle and on the pons and cerebellum. It is surprising that it did not cause symptoms corresponding to all these situations. There was no involvement of the third, fifth, sixth, seventh or eight nerve that could be determined. There was involvement, however, of the vestibular paths. This might readily be caused by pressure on the cerebellum. Of greatest significance was the hemiparesis. Hemiparesis always means something, it means a lesion on the other side of the brain. It is a localizing symptom of great value when taken in conjunction with Fröhlich's syndrome. I think the tumor shows that the chiasm really was not very much involved; the growth was separated from the chiasm, it was a little back from it. I can readily understand how the chiasm was not much pressed on by the tumor; hence the absence of hemianopsia. There was a papillitis, which some observers say is uncommon in pituitary cases. It was extremely marked in this case. The various symptoms, or lack of symptoms, resulting from such a large growth show how we might be misled in our diagnosis of these tumors. It was correctly diagnosed as a pituitary tumor, but this diagnosis was indicated more by the Fröhlich syndrome than by the localizing symptoms. The case illustrates, however, by the great size of the tumor, how these conditions might readily be confused with cases of pontile or cerebellar growths.

STUDIES IN DECEREBRATION *

II. AN ACUTE DECEREBRATE PREPARATION

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AND
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In an article entitled "A Method of Decerebration," [1] we described the technic of producing decerebrate preparations by rendering certain parts of the nervous system anemic. Briefly, this consisted of ligature of the basilar and carotid arteries. In this way that part of the brain cephalad to the ligature on the basilar artery may be made anemic. Such an occlusion of blood supply after fifteen minutes destroys the function of the anemic part permanently. By varying the level of the ligature on the basilar artery, various levels of decerebration can be obtained. It was pointed out that the method produced relatively sharply defined borders between normal and anemic or functionless parts, and therefore the study of function of certain levels of the central nervous system could be more accurately made.

Former methods of decerebration were mutilating in character and produced immeasurable degrees of injury about the level of the decerebration.

It has been stated that decerebrate rigidity follows transection of the brain stem, cephalad, between the anterior and posterior colliculi,[2] at the posterior end of the thalamus,[3] and at a plane bounded dorsally by the posterior limit of the anterior colliculi and ventrally by the region immediately caudad to the corpora quadrigemina.[4] It disappears when the section is made caudad to the plane of the entrance of the eighth cranial nerves and the calamus scriptorius has been reached. It follows that new observations must be made with methods producing sharper demarcations between functioning and functionless parts.

The preparation which is here described has been decerebrated by the anemic method at a level just cephalad to the exit of the fifth cranial nerve.

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* This article has been abbreviated by the omission of the illustrations, which appear in the reprints.

* Read before the Chicago Society of Internal Medicine, May, 1924.

1. Pollock, Lewis J., and Davis, Loyal E.: Studies in Decerebration. I. A Method of Decerebration, Arch. Neurol. & Psychiat. **10**:391-398 (Oct.) 1923.

2. Sherrington, C. S.: J. Physiol. **22**, Pt. 4, 319, 1898.

3. Thiele, T. H.: J. Physiol. **32**, Pt. 5 and 6, 358, 1905.

4. Magnus, R.: Arch. f. d. ges. Physiol. **159**:224, 1914.

About fifty animals (cats) have been decerebrated by this method. Without exception, decerebrate rigidity similar to that described by Sherrington has resulted. This rigidity varied as to onset and degree, depending on certain conditions, sequence of arteries tied, time elapsing between their occlusion, position and condition of the animal.

When the carotid arteries are occluded at the beginning of the experiment and the basilar tied later, rigidity ensues immediately. On the other hand, if the basilar artery is first ligated and immediately the common carotid arteries occluded, the rigidity slowly but progressively develops over a period usually varying from five to ten minutes. If the basilar artery is first tied and more than half an hour elapses before the carotid arteries are ligated, the assumption of rigidity will be immediate.

The operation is usually performed with the animal lying on its back. When the animal is permitted to retain this position following ligature of the artery, rigidity occurs more promptly and develops more rapidly than when the animal is allowed to lie on its side. In the latter instance, rigidity may begin early, but the marked extensor position of the legs is late in appearance, although the slightest attempt at flexion is followed by extensor thrusts, one succeeding the other until the legs are firmly outstretched.

The degree of rigidity becomes greatest when the animal is suspended in an upright position; as noted by Sherrington, antigravity positions, walking, running and standing, favor a more pronounced degree of extensor rigidity.

When fully developed, the rigidity is the same in whatever position the animal may have been placed. Of course, when suspended the degree of retraction of the occiput is less than when lying on the side.

An animal permitted to lie on its side without developing extensor rigidity following this operation will immediately develop complete extensor rigidity when it is suspended. Similarly, such an animal if turned on its back will develop the position immediately. Often there develops an immediate extensor rigidity, which after from three to four minutes subsides and is then followed by slower redevelopment of decerebrate rigidity. At times, there may be observed a vibratory tremor of the tail and the hind legs before the establishment of rigidity.

When, for any particular purpose, the carotid arteries are but clamped and after a period of less than fifteen minutes released, the decerebrate rigidity disappears. The subsequent occlusion of these vessels is immediately followed by marked decerebrate rigidity.

The general condition of the animal markedly influences the onset of the rigidity. If, for some reason, as shock, prolonged anesthetic, or hemorrhage, the animal is in poor general condition, decerebrate rigidity is delayed in development, and it does not attain its usual proportions.

Such operative complications should not and do not occur after the technic has been acquired. When fully developed, the rigidity is marked, the spine is extended, the tail is held in a horizontal position or is curved strongly upward, at times to the degree of lying parallel to the back. Any attempt at depressing the tail is followed by immediate resumption of the original position. The extremities are fully outstretched, the forelegs are held quite stiffly extended at the shoulder and elbow joints and retracted. The angles between the limbs and the body vary as to the position of the body; if the animal is placed as if sitting on its haunches, the angle is smaller than if the trunk is horizontal. The wrist joints are seldom involved in the rigidity. The hind legs thrust backward are outstretched, but to a lesser degree. All the joints in the hind legs are equally involved in the rigidity. The head is maintained in a lifted position despite gravity, and there may be marked opisthotonos of the head on the neck. The degree of opisthotonos may be increased by pressure against the outstretched fore limbs or by moving them forward. The jaws are tightly closed. Considerable force is required to produce flexion of the extended extremities, and when released they immediately assume their former position.

Such an animal, if carefully posed, can be made to stand. Numerous pseudo-affective and other reflex reactions may be demonstrated.

Often, when soon after tying the basilar and carotid arteries the animal is placed on the floor on its belly, it will turn its head to one or the other side, and if the head should be turned to the right, the right fore leg will be flexed and the left fore leg strongly extended in front of the animal. It will then, by moving fore and hind legs aimlessly, progress, generally in a semicircle. When an animal is suspended, immediately after ligature of the vessels and rigidity is slow in development, there will be observed movements which are definitely of a character of "walking," alternate movements of all four legs, contralateral as to fore and hind legs. Such movements are ordinarily interpreted as automatic associated movements. Cobb [5] observed that these movements were lost when a transection was made just cephalad to the red nucleus, and Mella [6] showed that in cats there is an area in the subthalamic region, removal of which causes cessation of the automatic locomotor movements associating the fore and hind legs. The occurrence of these movements in our animals stresses the point mentioned before, that mutilating operations cannot determine the levels of function in the central nervous system.

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5. Cobb, Stanley; Bailey, G. G., and Holtz, R. R.: Am. J. Physiol. **4**:239, 1917.

6. Mella, Hugo: The Diencephalic Centers Controlling Associated Locomotor Movements: An Experimental Research, Arch. Neurol. & Psychiat. **10**: 141 (Aug.) 1923.

When such movements have developed, they may be stopped by gradually lowering the animal until the fore legs firmly touch the table above which they were suspended. They may likewise be stopped by grasping the extremities, gradually molding them into an extended position. The slightest stimulus or change of position reestablishes them. If the hind legs alone are lifted from the table, the movements occur only in these legs. At times, when the movements have been stopped by lowering the animal to a standing position, pinching or stroking the animal's legs starts the walking movement.

These movements do not occur when the animal lies on its back, and they are not typical when the animal lies on its side. They are then more aimless and irregular.

After the complete development of rigidity, these movements diminish and finally disappear. In animals developing marked rigidity immediately, they are not constantly present. It would appear that the more marked the rigidity, the less do these movements present themselves. The explanation may be found in the observation of Bremer [7] that a certain optimum tonus is necessary for the production of rhythmic progressive movements. As rigidity increases, the optimum tonus for progressive reflexes is passed, and they disappear.

Certain reflexes may be elicited immediately after conclusion of the operation, testifying to the small amount of shock which results. Stimulation of the cornea produces winking and a nictitating reflex. Stimulation of the nostrils elicits sneezing, shaking of the head and at times contractions of the abdominal and perineal muscles. Stroking the soft palate is followed by inspiration. Swallowing and licking may follow the production of sialorrhea; for example, by the injection of picrotoxin. Vibrissae and pinna reflexes occur. Stimulating the auditory canal will produce a shaking of the head, movement of the ear and a scratch movement of the ipsolateral hind leg. At times, this scratch movement may be evoked from stimulation about the mouth, nose and throat, and occasionally it may seemingly occur spontaneously. When this occurs, the opposite hind leg becomes flexed. Anal stimuli produce perineal contractions. Apparently spontaneous twitching of the ears and winking occur. Pinching the hind foot produces a flexor withdrawal rarely associated with flexion of the opposite hind leg, and a few clonic movements. Extensor thrusts are readily evoked. The pupils are widely dilated, and the light and ciliospinal reflexes disappear. The animal is apparently blind, oblivious to other animals in close proximity. The animal does not react to auditory stimuli, although the decerebration is made above the level of the eighth pair of cranial nerves. No

7. Bremer, Frederick: Arch. Internat. de Physiol. **9**:189, 1922, quoted by Bailey, P.: Arch. Neurol. & Psychiat. **10**:152 (Aug.) 1923.

mewing has been noted, although at times a sound accompanies strong expiratory efforts. The body temperature of these animals does not immediately diminish, but usually there is a gradual decrease to the time of death. Some animals have a rectal temperature of 35.5 C. five hours after decerebration. In fact, one animal had a temperature of 41 C. six and a half hours after decerebration. The animals are easily overheated, and the use of the heating pad on which the animal lies raises the temperature rapidly. When the pad is removed, the body temperature does not fall, but often rises to a degree of hyperthermia sufficient to produce death.

Protocol for Cat 32

10:10 a. m.....37.8C.	12:14 p. m.....36.7C.
10:40 a. m.....36.7C.	1:35 p. m.....36.9C.
11:05 a. m.....36C.	2:22 p. m.....36C.
11:15 a. m.....36C.	3:08 p. m.....35.3C.
11:45 a. m.....35.4C.	3:30 p. m.....35.1C.
12:00 m. .....35.5C.; (Heating pad applied)	5:40 p. m.....34.8C.
	9:00 p. m.....33.5C.

The poikilothermic animals frequently produced by mutilating decerebration differ from these probably because of the extensive trauma and shock in mutilating operations. Further work must be done as the decerebration in our animals is far caudad to the hypothalamic region.

The decerebration does not interfere with regular rhythmic respiration. Under proper conditions of anesthesia and the absence of hemorrhage, the anemic decerebration produces no respiratory difficulties even immediately following this operation. When the animal is in poor condition, or overanesthetized or is suffering from hemorrhage, temporary respiratory difficulty and irregularity is observed.

When a hyperpnea has been produced by picrotoxin, anemic decerebration is immediately followed by the assumption of regular respiration and a normal rate. Apparently the effect of the picrotoxin is expended on the higher respiratory centers in the colliculi.

The pulse rate is changed no more than by any operative procedure. Involuntary urination is a constant accompaniment of the production of full decerebrate rigidity. Vomiting is not produced by the decerebration.

Tonic reflex phenomena or standing reflexes described by Magnus and DeKleijn [8] are demonstrable both in the relations of the head in space and to the rest of the body, that is, from stimulation of the otolith organs of the labyrinths and the neck muscles. If the head be rotated so that the chin points to the right, vertex to the left, the

8. Magnus and DeKleijn: Arch. f. d. ges. Physiol. **159**:224, 1914; **163**:405, 1916.

extensor rigidity in the right fore and hind legs is distinctly increased, while the left fore and hind legs become more flexed. If the head be rotated back to the midline, this inequality disappears. If the head be turned so that the chin points to the left, the rigidity increases in the left fore and hind legs and is much decreased on the right side; in fact, actual flexion may occur. These changes persist as long as the head is held in the abnormal position. Retraction of the neck on the head is accompanied by a decided increase in the degree of extensor rigidity both in the fore and hind legs. In addition, the fore legs assume a position with less retraction at the shoulder joints. When these animals are allowed to lie on their backs with their heads in the median position, all four extremities are held equally in extensor rigidity. If the animals are placed on one side or the other, the rigidity is more marked in both extremities on the side on which it is lying. The head tends to assume a position of opisthotonos when the animals are lying on their sides more easily and persistently than when they are suspended.

Contrary to the observations of Weed,[9] Sherrington,[2] Thiele,[3] and Horsley [10] have all found that the extirpation of the cerebellum does not abolish decerebrate rigidity.[4] Magnus has established beyond any doubt that decerebrate rigidity persists undiminished after removal of the cerebellum and bilateral division of the eighth nerves.

In our former communication, we noted that in this preparation the superior half of the cerebellum is included in the anemic area. Neither in this preparation nor in one in which the ligature was placed on the basilar artery at the junction of the vertebral arteries below the posterior inferior cerebellar artery, producing a decerebello-decerebrate preparation, was the decerebrate rigidity in any way affected.

The animals have stood subsequent abdominal and other operations exceedingly well.

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9. Weed, L. H.: J. Physiol. **48,** Pts. 2 and 3, 205, 1924.
10. Horsley, Victor: J. Physiol. **29,** Pt. 6, 446, 1906.

THE SIGNIFICANCE OF CERTAIN FACTORS FOR THE DEVELOPMENT OF THE HEREDOFAMILIAL DISEASES OF THE NERVOUS SYSTEM

(CONSANGUINITY, RACE, ENDOCRINOPATHIES, TOXINS, INFECTIONS, ALCOHOLISM, ROENTGEN RAYS)

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The newer studies of heredity have thrown some light on the relations of certain factors (such as those mentioned in the title) to the occurrence, or to the development, of the so-called heredofamilial diseases of the nervous system (limiting this term to diseases that occur in families because of their idiotypic or genotypic constitution). The patients who suffer from heredofamilial diseases of the nervous system become pathologic phenotypes, because of the idioplasm or germ plasm, that has resulted from the fusion of their parental gametes. By one parent, or by both, hereditary factors (ids or genes) responsible for the occurrence of the disease must have been transmitted. I shall discuss briefly the present state of knowledge concerning the significance of consanguinity, race and certain other influences for the development of the types of disease that are under consideration.

CONSANGUINITY

Biologists have long been interested in the origin of the taboo of marriages among those of close blood relationship and of the special loathsomeness that attaches to the idea of sexual commerce of near kindred (incest). We now know that the strong sentiments that exist against close intermarriage and against incestuous relations, not only among civilized people but also among the more primitive races, are biologically well founded, since undesirable traits due to recessive factors in the idioplasma tend to appear in the offspring of cousin marriages, whereas they tend to disappear in the descendants of parents who are not of the same blood (so-called "outmatings").

The degree of blood relationship popularly regarded as "consanguinity" is that of first or second cousinship and of all still nearer kinship. But according to modern views of heredity, this popular idea is too narrowly conceived, since the same ancestral traits, it is now believed, may be carried by fifth, or even by tenth, cousins. The more distant blood relationships can scarcely be escaped, of course, in any marriage, for all people of European origin are probably as closely related, it has been estimated, as thirtieth cousins. Marriages of close

consanguinity are favored by obstacles to free and wide selection of mates, as is evidenced by the populations of small islands, of peninsulas and of secluded mountain valleys, or by groups of people in which social conditions (clanship, race, institutional life and membership in small religious sects) have acted as barriers to free marital selection.

Among the abnormal conditions of the nervous system, the occurrence of which in the offspring is known to be favored by consanguineous marriage when the stock is correspondingly tainted, are: (1) hereditary spastic paralysis, (2) congenital deaf-mutism, (3) retinitis pigmentosa, (4) myotonia congenita, (5) imbecility and (6) certain forms of insanity.

It is, however, only when the idioplasm of the family stock is tainted that there need be apprehension for the offspring of the closely related mates. Determiners of traits are not produced anew by consanguineous marriages; these merely give the opportunity for determiners already present to reveal themselves in offspring by manifestations that may not have appeared in the parents. Consanguineous marriages entail no danger to the offspring if the idioplasms of the stocks are good; on the contrary, there is reason to believe that the special gifts met with in good strains without taint would tend to reappear in the descendants of parents who are cousins.

Much that was puzzling with regard to the mysterious effects of consanguineous mating or "inbreeding" has been made clear through mendelian studies of the so-called recessive or latent mode of heredity. Students of mendelian heredity have concluded that for each unit character there is in the idioplasma of the sex cells (ova and sperm cells) before they undergo reduction-division not a single id or determiner, but a *pair* of such ids. When such sex cells (diploid) undergo reduction-division (half the parental idioplasma of each cell being thrown out) to form the gametes (haploid), only one of the two members of each pair of ids representing a unit character is retained in the gamete. When a male gamete fuses with a female gamete to form a zygote (fertilized egg cell), a diploid cell results, and the characters that develop in the new creature will depend on the contents of the new pairs of ids that have been formed; a so-called "dominant" character will become manifest in the phenotype if its corresponding id has been received into the idioplasma from either the male gamete alone or from the female gamete alone (heterozygote state) or from both gametes (homozygote state), whereas a so-called "recessive" character cannot become manifest if a corresponding id has come from only one gamete and not from the other (heterozygote state), since for the manifestation of this character in the phenotype it is essential that its corresponding id must be received into the idioplasma of the zygote from the gametes of both parents (homozygote state).

If the view mentioned be valid, it will be clear that a heredofamilial disease dependent on a hereditary factor that is dominant, for example, neurofibromatosis, may be nearly as prevalent among the offspring of outmatings as of inbreedings; but it will also be clear that in a heredofamilial disease in which the hereditary factor remains recessive on heterozygote pairing of its id and can become manifest only on homozygote pairing of the id, the opportunities for the entrance of such a rare id into a homozygote pair that can determine the manifestation of the disease in its bearer must be far greater among the fertilizations of consanguineous marriages than among those of marriages of unrelated persons. Great numbers of persons through several generations might serve as carriers (conductors) of such ids of a recessive character and yet have offspring that remain free from the disease, for only when a conductor of a rare id happens to mate with another such conductor is the opportunity provided for the combination of two of the same kind of rare ids to form the pair of similars that alone can compel a recessive character to become manifest.[1]

Although the explanation of the special tendency of recessive hereditary types of familial diseases to become manifest among the offspring of consanguineous parents contributed much to our understanding of the appearance of the disease in descendants (despite the skipping of one or more generations), there still remained to be answered the question: Why is it that for some recessive heredofamilial diseases statistical studies indicate a persistently low incidence of close parental blood relationship (e. g., only 2.2 per cent. for dementia praecox) whereas for others a persistently high incidence has been found (e. g., 16 per cent. in retinitis pigmentosa)? An apparently satisfactory answer to this question has been made by F. Lenz, who has suggested that among the recessive diseases the rarer the incidence of the disease

1. The conception here outlined seems to me to accord best with the known facts of the recessive heredity of certain of the familial diseases of the nervous system. There are some biologists who look on inherited characters as developing from single determiners rather than from pairs of such determiners; they explain the recessive heredity of certain nervous diseases by assuming an absence of the single specific determiner, an explanation that will be found to be in general less satisfying, especially when the phenomena of reduction division are considered, than that which assumes a pair of determiners in the idioplasma as the basis of each hereditary unit character.

It should be borne in mind, furthermore, that some hereditary diseases cannot be accounted for by the assumption of a single id or pair of ids, but appear to depend on several pairs of ids (*polyidia*); also that possibly sometimes one pair of ids may influence the development of several different phenotypic traits (polyphænic heredity).

In order to keep the presentation simple, no mention has been made of *epistasis*, in which one id suppresses the determining power of another id that belongs to a different pair of ids, nor of *hypostasis*, the opposite condition.

itself the greater should be the incidence of blood relationship among the parents of persons suffering from it. For it would seem to be clear that the less common the latent id carried by a "conductor," the greater the improbability of his finding a marital partner carrying the same latent id among persons other than his own blood relatives.

RACE

The term "race" is not easy to define, although in general we mean by it a large branch of the human race consisting of an aggregate of persons that resemble one another more or less closely because of some limitation through a long period of time of matings with members of other large branches of the human race. The three groups of men, (white, yellow, and black) are divisible into many subdivisions, although there seems to be no general agreement among ethnologists as to the best classification of them. Among the subdivisions of European man, the Teutonic (or Nordic), the Alpine and the Dinaric races are multiply subdivisible. Pure race lines in the mendelian sense are unknown among human beings who in mendelian terminology must be designated as polyhybrid heterozygous bastards. What we are concerned with is the problem of the existence or nonexistence of any special relationship of race (or of race admixtures) to the heredofamilial diseases of the nervous system. In no country in the world, probably, are so many races and subraces (English, Scotch, Irish, French, Germans, Hollanders, Scandinavians, Austro-Hungarians, Hebrews, Italians, Poles, Portuguese, Greeks, Japanese, Chinese, Negroes and Indians) so liberally represented as in the United States of America. Race and language often act temporarily as obstacles to free selection in the marriage of immigrants, but sooner or later there occur many racial admixtures ("alloys"). The sooner the keeping of personal and family records of phenotypic characters becomes common, the better for advances in our knowledge of the significance of racial influences for the occurrence of various diseases. The type of genealogic work conducted by Davenport and his associates of the Eugenic Record Office is worthy of widespread imitation.

The incidence of amaurotic familial idiocy among Polish Jews hints at the importance of race in etiology. The recent attempts of the students of the pathology of constitution to correlate psychophysical types with race forms are also interesting, although the suggestion of Stern-Piper [2] that we look on the asthenic constitution (habitus asthenicus) as a Nordic type associated with a schizoid tendency and on the pyknotic constitution (habitus arthriticus, habitus apoplecticus) as an

2. Stern-Piper, L.: Kretschmer's psychophysische Typen und die Rassenformen in Deutschland, Schweiz. Arch. f. Neurol. u. Psychiat. **67**:569, 1923.

Alpine type associated with a cycloid trend will probably be startling to even the credulous.

ENDOCRINOPATHIES

Experimental studies on the influence of endocrine products (increta) on growth in early stages of development (for example, in the larvae of amphibians) make it seem probable that in the developmental processes of both normal and abnormal phenotypes the internal secretions may play an important rôle. Again, the profound neural disturbances observable in certain diseases of the endocrine organs emphasize the significance of the incretions for the behavior of the neural and psychic functions; I need mention only the psychic and autonomic phenomena noticeable in exophthalmic goiter and in myxedema, the myasthenia that may be associated with thymic disease, the tonic spasms of tetany in parathyroid insufficiency, the peripheral neuritis that may accompany diabetes mellitus due to deficiency of pancreatic production of insulin, and the profound alterations in the psyche that occur with changes in the internal secretions of the gonads at puberty and at the climacteric, to illustrate what I mean. The chemical coordination and regulation of the body, the behavior of the living organism as a whole as a functional unit, must in no small part depend on the activities of the incretory organs. The conclusion is almost unavoidable that the ids, or determiners, of hereditary characters must, to a considerable extent at least, be operative through the intermediation of these endocrine organs in the production of varieties among phenotypes, including those pathologic personal variants that are victims of the heredofamilial diseases. Plausible as are these deductions from theory, it must be admitted that but little more than a start has been made in the direction of collecting data that bear on the relation of endocrine disorders to heredofamilial diseases; but the field has been opened, and it invites intensive cultivation. The reduction of cretinistic imbecility in Aosta brought about through segregating the cretins and preventing them from marrying (Jordan[3]), the demonstration of recessive heredity in a glycosuric family (Bramwell,[4] 1908), of a dominant heredity in a family exhibiting diabetes insipidus (Gossage,[5] 1907) and of a dominant, partial sex-limited heredity for the exophthalmic goiter diathesis that may account for the preponderance of incidence of

3. Jordan, D. S.: Cretinism in Aosta, Eugenics Rev. **2**:247-248, 1910.

4. Bramwell, B.: Diabetes Mellitus; Strong Hereditary History; Differential Diagnosis of Glycosuria and Diabetes Mellitus, Clin. Studies, Edinburgh **6**:263-266, 1907-1908.

5. Gossage, A. M.: The Inheritance of Certain Human Abnormalities, Quart. J. Med., Oxford **1**:331-347, 1907-1908.

exophthalmic goiter in females (Lenz,[6] 1917), illustrate a growing interest in the reciprocal relations of the endocrinopathies and abnormal idiotypic constitutions.

TOXINS, INFECTIONS, ALCOHOLISM AND ROENTGEN RAYS

Much speculation has been indulged in with regard to injurious chemical and physical influences of exogenous origin as causes of heredofamilial disorders. In acting on persons derived from tainted stock, such agents could act only as "releasing factors" or "accelerating factors" to which certain persons react in a pathologic way because of their inherited tendencies. That such exogenous influences reaching germ cells within the bodies of persons who later become parents may alter the idioplasma (idiokinesis) so as to give rise to mutations (idiovariations) that are the starting point of heredofamilial disorders, is conceivable and even to be regarded as probable in the light of the work of biologic experimenters.

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6. Lenz, F.: Ein Beitrag zur Kenntnis der Basedowdiathese, München. med. Wchnschr. **64**:292-294, 1917.

Clinical and Occasional Notes

APPARATUS FOR SIMULTANEOUS DISPLACEMENT

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DESIGNED FOR THE SIMULTANEOUS DISPLACEMENT OF FLUIDS IN THE SPINE, JOINTS OR BODY CAVITIES, BY AIR OR OTHER GAS

With a view to making a more extensive application of the method of diagnosis in brain and cord lesions by means of radiography, after the displacement of cerebrospinal fluid by air, and also for the purpose of preventing discomfort or untoward effects on the patient as a result of such procedure, the author was stimulated to devise the apparatus described below.

This apparatus may be used not only for the displacement of spinal fluid by the lumbar route, but also for the displacement of fluids in joints, or body cavities, as in certain pleural effusions and ascites.

Essentially the apparatus consists of a small bore cannula placed within another cannula of larger bore; the two are connected to one cylinder; the larger bore cannula is for the passage of the fluid, and the smaller is for the simultaneous entrance of air to displace the fluid withdrawn. It is, of course, understood that the size of the containers to be used must be commensurate with the cavity involved, and the quantity of fluid to be displaced. *This description is confined to the use of the apparatus in the displacement of spinal fluid.*

PURPOSE OF THE APPARATUS

The purpose of the apparatus is:

1. To permit the displacement of spinal fluid by air without alteration in the intracranial and intraspinal pressure during the operation.

2. To permit with more safety the removal of spinal fluid in cases of posterior fossa tumor.

3. To permit replacement of the withdrawn cerebrospinal fluid after the latter is found to be normal, and thus avoid the possible distress due to the expansion of the air within the ventricles or their irritation by the same. Also to prevent the dizziness that the patient experiences from the succussion of the residual fluid in the partly filled ventricles.

4. To prevent haste in removing the cerebrospinal fluid, or forcing in a quantity of air above that which can be disseminated in the ventriculosubarachnoid system.

HOW THE APPARATUS FULFILS THE PURPOSE FOR WHICH IT WAS CONSTRUCTED

1. The apparatus is so constructed that it forms another loop in the ventriculosubarachnoid system and is (during the exchange of the fluid) really an integral part of the system. All the air that will be introduced into the cerebrospinal system is found in this loop. All the operator needs to do in order to displace by air the fluid in the cranial and spinal cavities is to alter the relative level of the loop with regard to the position of the patient. This is done under constant manometric control.

2. In using the apparatus, ordinarily, only from 2 to 4 c.c. of fluid can be removed without the entrance of an equal amount of air to displace it, and in that way the intraspinal, and consequently the intracranial, pressure is equalized.

3. The apparatus permits shutting off of the loop during the radiographic procedure, and if radiography proves negative, the cerebrospinal fluid is made to displace the air by reversing the relative levels of the container and the patient.

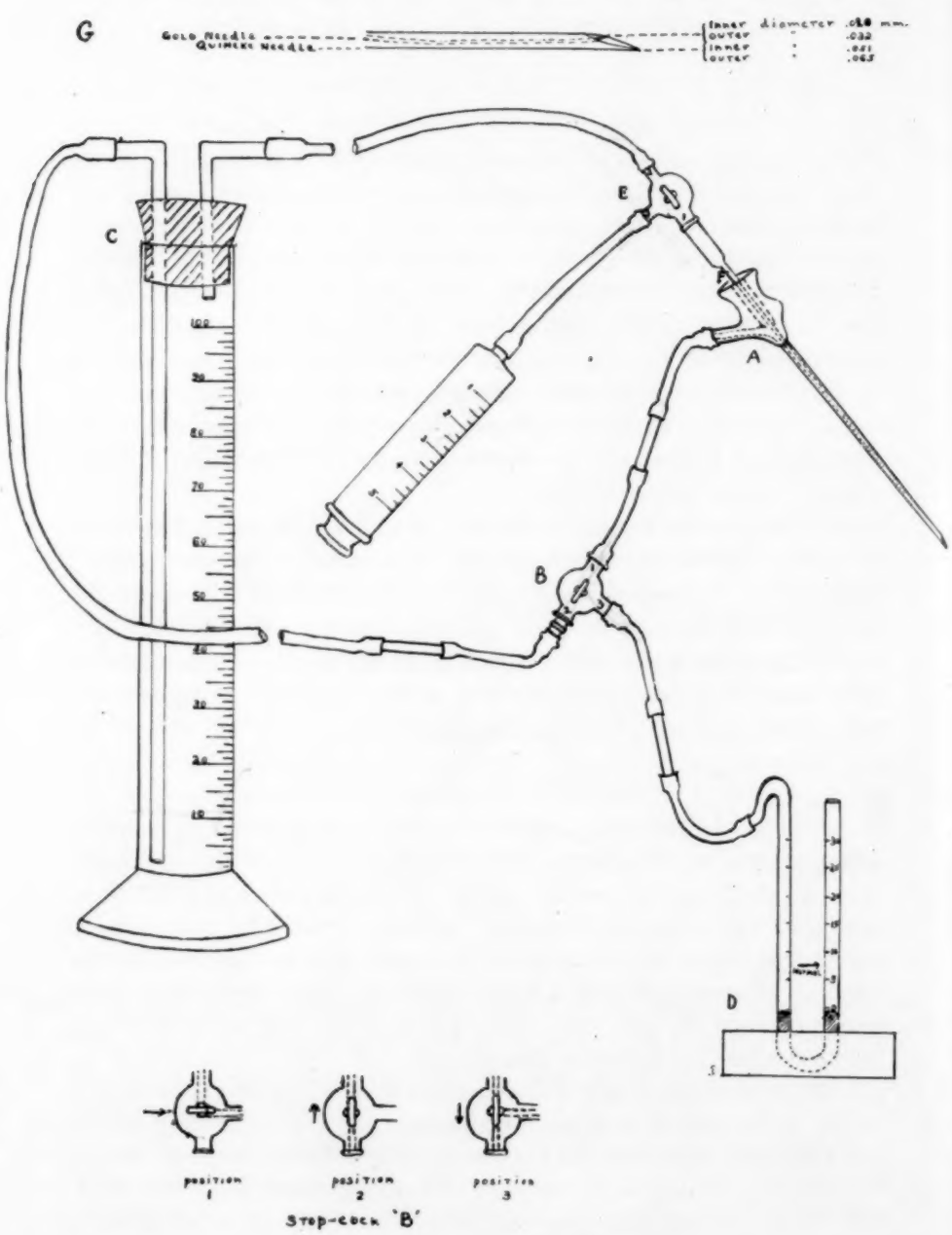
4. The diameters of the two needles of the spinal apparatus are so constructed as to permit only from 5 to 10 c.c. of fluid to be removed per minute; or none, if no air takes its place.

DESCRIPTION OF THE APPARATUS

As stated in the foregoing, the apparatus is essentially a cannula within a cannula. The larger bore cannula is of the ordinary Quincke type of spinal puncture needle with an internal diameter of 0.051 inch, having an extra tube attachment in the side of the hub. This tube is placed just behind the junction of the needle with its hub, so that the hub can be stoppered with a long stopper and still permit fluid to be drawn through the side tube. This will be made clear by reference to the sketch and the following description:

The tube of the needle *A* is connected by a 6 inch length of rubber tubing to one end of a three-way stopcock (*B, 1*). The other end of this three-way stopcock (*B, 2*) is connected by a 30 inch length of tubing to a long glass tube in an air-tight 100 c.c. graduated cylinder (*C*). The third end of the three-way stopcock (*B, 3*) is attached to a manometer.

The inner needle (F) is made of very thin gold tubing, and has an external diameter of 0.032 inch. (When the apparatus is used for tap-



Simultaneous displacement apparatus.

ping of the chest or abdomen, larger containers as well as larger cannulas are used.) Its wall is only 0.006 inch thick and is slightly curved, so that its tip extends beyond the beveled end of the lumbar puncture needle when the former is placed within the latter (*G*). The hub of this needle is a three-way stopcock; one end of the stopcock (*E, 2*), is connected by a 36 inch length of rubber tubing to a short glass tube in the stopper of the 100 c.c. cylinder (*C*). Another outlet of the stopcock (*E, 3*) is connected to a 5 c.c. air-tight glass syringe. The third outlet (*E, 1*) of the stopcock continues as the inner needle (*F*).

Glass insert tubes are used as connectors in order to facilitate observation. All connections must be absolutely air-tight. Rubber tubing with a thick wall is used.

METHOD OF USE

The apparatus is sterilized in sections; the stoppered container, the rubber tubing and stopcock, and the two needles with their stylets separately. (Before the apparatus is assembled, care must be taken that the rubber tubing is drained of its fluid). The lumbar puncture needle is introduced between the spines, pointing slightly upward so that the hub end of the needle is at least 1 inch lower than its sharp end. The patient is either reclining on his side or is sitting.

The rubber tubing that was already connected to the long glass tubing of the cylinder is now joined to the metal tube (*A*) of the lumbar puncture needle; the stylet of the lumbar puncture needle is then withdrawn and a few drops of fluid are permitted to be taken for cell count. Immediately after the removal of the stylet, the smaller needle is introduced in its place, care being taken that the hub of the smaller needle fits tightly within the hub of the spinal puncture needle and is air-tight. (A drop of sterile petrolatum will usually suffice to insure this.)

When the stopcocks are closed, a pressure reading is made. Five cubic centimeters of air is drawn from the cylinder into the syringe, and then the stopcock is reversed and the air is gently fed into the spinal canal. The entrance of air can be definitely ascertained by placing a stethoscope over the lumbar spine, and if a gurgling or trickling sound is heard, one is definitely certain that the air is entering the spinal canal.

As soon as this gurgling sound is heard, the stopcocks are opened to position 2; that is, both needles are in connection with the cylinder. The cylinder is lowered and adjusted to such a level that the displacement of the fluid will occur at the rate of 5 c.c. per minute. As each 10 c.c. of fluid are withdrawn, the stethoscope is placed against the spine to ascertain that the exchange of air for fluid is going on, also a manometric reading is made.

After 40 or 50 c.c. of fluid have been displaced, the patient's head is slightly rotated in such a way as to get the fluid out of dependent spaces. The exchange is continued until air is seen continuously in the glass insert of the fluid cannula. The stopcocks are closed to the spinal canal, and the necessary roentgenograms are taken; if no abnormality is found the stopcocks are opened and the cylinder is raised above the level of the patient, and the patient's head is lowered below the level of the spine. In this way, the air is permitted to reach or to ascend into the cylinder and the fluid permitted again to take its place in the cerebrospinal system.

Note that the cylinder is kept at about body temperature during the period of collection and replacement. In case the fluid shows a high cell count or turbidity, and it is desired to replace the spinal fluid with some medication, the medicated fluid should be placed in a similar cylinder, using the same quantity as that which is found in the cylinder with spinal fluid. The cylinder with the medicated fluid is substituted, and before replacement is started enough spinal fluid is to be permitted to enter into the long glass tube of the cylinder so that suction may be started; otherwise it would be impossible to displace the air with the medicated fluid.

Under no circumstances should one draw fluid from the cannula by means of a syringe, nor should one draw fluid in case the gurgling sound is not heard over the lumbar vertebra at least one space higher than that of the lumbar puncture.

CLINICAL NOTE

So far, only fifteen cases have been subjected to this method of spinal fluid displacement and ventriculography. Some of the patients were shown to be normal, others to have retractions of the brain (general paralysis); one had brain tumor; one patient had a fracture of the skull (which did not show otherwise); and one had a spinal tumor.

The roentgenograms compare well with those taken after the introduction of air by the direct ventricular route. The patients stood the procedure much better, especially patients with general paralysis who have shown improvement from day to day; some of them have even been restored to useful pursuits in life. In only one of these cases was there observed any rise of temperature (1 C.), and in that case the spinal fluid displacement and ventriculography were performed six hours after an injection of arsphenamin. The other cases showed no other effects than those observed in ordinary spinal punctures. One patient who had a systolic blood pressure of 240 showed no unusual reaction to the procedure.

111 East 87th Street.

ASTEREOGNOSIS*

FOSTER KENNEDY, M.D., NEW YORK

A discussion almost turbulent in its tenseness was evoked last June at the meeting of the American Neurological Association concerning a case demonstrated by Patterson Ross from Cushing's service as an example of astereognosis of cerebellar origin. It was plain from the trend of remarks made on that occasion that clarity of outline has not yet been achieved in this minor subject.

It might be well to review—albeit from memory—the case in question, from which a general discussion of the subject may be developed.

A middle-aged man presented ingravescent signs of increase in general intracranial tension. He had headache, vomiting and bilateral papilledema. He was unable by feeling to name or recognize objects placed in his left hand, the movements of which were at the same time somewhat unsteady.

Because of these signs, it was thought that the right postrolandic area was the seat of the tumor. This zone was accordingly exposed, and nothing was found.

Later, there developed nystagmus of coarse quality on looking to the left; the ataxia of the left upper extremity grew grossly hypermetric; and the patient reeled constantly to the left side in walking. The astereognosis previously noticed in the left hand persisted. No gross sensory defects of other forms were found in the arm or hand.

The infratentorial region was operated on, and a left cerebellar tumor was exposed and removed.

The contention that this case proved that astereognosis could exist as a sign of cerebellar defect excited annoyed denial from many upholders of physiologic orthodoxy.

Through Dr. Cushing's kindness I was enabled at leisure to examine his patient with Dr. Ross the next morning, when it was evident that vibration sense was quite abolished in the left arm and hand, and that deep pain also was definitely diminished in its muscles. One mistake in five trials was made in the sense of position of the fingers.

In short, by the tumor in the left cerebellar lobe there had been exerted a noxious pressure on the left arm afferent fibers of the posterior columns or their nuclei, and a strictly localized high tabes had been produced—a simple enough explanation of the mystery.

None the less was it plain that the word astereognosis to different competent men meant slightly different things, but that the faculty or sense so named was thought by most to have a quite definite topographical setting.

First, what is the current clinical test in this matter? Is it not to place in the suspected hand various familiar objects and ask the patient to recognize and name them without the use of the eyes? Inability on the part of the patient to do this correctly may of course be due to many different defects: motor, sensory, psychic or linguistic; failure from the patient's point of view may be complete, yet all would agree that astereognosis—meaning sensory loss—would not be present were the hand palsied or anesthetic, though in the sense of faculty loss its presence would be clear. My contention then in this short communication is simply this: that stereognosis is a faculty—in Shakespeare's words—compounded of strange essences—as distinguished from a pure

* Read before the New York Neurological Society, March 4, 1924.

and simple sensation. To make our meaning clear in clinical examination and reporting, it is therefore necessary to elaborate or qualify the word by an annectant phrase indicating the cause of the loss of the faculty in question.

The word literally only means the power to recognize the solidity of objects, or more precisely their dimensional qualities—but writers such as Tilney and Riley expand its meaning to include knowledge of weight and consistency as well, and in our daily work we throw our net still wider.

For the accurate performance of the common test just described, there must be present adequate motor power, accurate superficial sensibility, accurate deep sensation, accurate kinesthetic memory pictures capable of ready facilitation to consciousness, an intact associative system to wherever we store our visual images, and an association system in good working order to our temporal convolutions to ensure the proper naming of the object whose dimensional characteristics have been appraised, discriminated, classified and psychically seen. Clearly then the loss of the faculty of stereognosis may occur through the absence of any one of its component ingredients, just as a knee jerk may be abolished by the collapse of a posterior root, a ventral horn or a mixed peripheral nerve.

It has been shown by Head[1] that the affective center of pain, touch, hot and cold sensations is in the optic thalamus, and that the cerebral cortex in its so-called sensory zone presides over only discrimination and spatial projection. On the other hand, these functions are precisely those disturbed through interference with the posterior spinal columns; a patient with high tabes, a cervical Brown-Séquard paralysis with motor recovery, a subacute combined sclerosis of the upper cord, and certain cases of multiple sclerosis, all fail to carry out three simple dimensional tests, as did the patient discussed in the beginning of this article. It is therefore clear that spatial perception, dimensional appreciation and, in fine, the mechanism for performing our simple clinical tests for "astereognosis" can be destroyed at any one of many different functional levels, and several different anatomic levels as well. The compass test or discrimination of two simultaneously made contacts is futile in the presence of tactile anesthesia; it is disturbed in gross posterior column disease, and only becomes of value as an indicator of cortical integrity in the proved health of the afferent system up to and including the essential center of the thalamus.

Astereognosis therefore must be described as depending either on peripheral afferent defect, or on lesions of the spinal afferent system, either of tactile or, as is more usual, of posterior tracts; or on a thalamic lesion, or on cortical or subcortical abnormality.

Clearly, it is the last named which we usually mean when we discuss astereognosis, but its presence in my experience cannot be proved when deep sensory impulses are quite uninterrupted, and visual and speech integrations are intact. We must stress also the psychic process involved in this function or faculty. The cortex is the repository of past impressions and sensory impulses arriving there which must be discriminated and classified according to the kinesthetic memories already stored by experience; Oppenheim[2] explains the frequency of astereognosis in infantile hemiplegia by the fact that no tactile memory pictures have been acquired by means of the paralyzed hand.

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1. Head: Studies in Neurology **2**:676.

2. Oppenheim: Text-Book of Nervous Disease, Chicago, Chicago Medical Book Co. **2**:704.

Our tests in this matter are entirely too gross and result from some confusion of thought on our part—we fail to distinguish in our examination between form description and recognition, and object description and recognition, nor are we always sufficiently careful to exclude the possibility of there being some impairment in subsidiary sense impressions.

Even as a result of purely cortical or subcortical postrolandic lesions, if we examine our patients with enough diligence we shall find a quantitative reduction in kinesthetic sensibility and in the delicacy of the compass test. Vibration sense in such cases is usually but little affected. We must have, therefore, as a basis for astereognosis, a deficient supply of sensory information —judgment is necessarily impaired, for deductions can no more be formed without premises than can bricks without straw—if then spatial and kinesthetic sensory information be sufficiently lacking, the patient will be unable to achieve form recognition, and not being able to achieve form recognition, he will be without the selecting stimulus to appraise his visual memories correctly and will *a fortiori* fail in object recognition.

Our demand for object recognition is less a test of sense than of an abstruse psychic association, and failure even to achieve recognition of form—a loss of function rather than sensation—depends fundamentally on deprivation of adequate information through sensory defect; this defect may be cortical in site, thalamic, spinal, or peripheral. The latter is simple to discover, the former more difficult, but with caution, possible.

Abstracts from Current Literature

APHASIA AND THE ARCHITECTONIC DATA. LUIGI RONCORONI, Riv. di patol. nerv. **28**:17, 1924.

Pathologic anatomy cannot completely explain the pathogenesis of aphasia if it is limited simply to the localization of the alterations in the convolutions of the cortex or in the subcortical areas. An enormous amount of work done with mathematical exactness is necessary to complete the pathologic studies in cases of aphasia. The author has in mind architectonic studies in various areas of the cortex. No discussion of this subject is definitely complete without such studies. Occasionally, single cases of aphasia may fit in with a particular conception, but they must be considered as incompletely studied without minute architectonic research. The cerebral cortex must be considered as a synthesis of various organs which do not have an exact correspondence with gyri and convolutions. The various areas in the cortex subserve certain elementary functions which by themselves are simply one phase of many psychic activities. These elementary functions of the various areas of the cortex consist of sensations and memories. When these are synthesized by a series of successive integrations, the highest types of psychic function become possible. The personality, therefore, must be considered as a complete integration of all these integrated functions. Cortical areas are distinct only in primitive animals in which only the common elementary functions are elaborated; for example, the striate areas of the occipital cortex whose sole function is the perception of light. There are evolutive areas which have the function of externalizing elementary functions peculiar to the species, such as those of speech in man. The cortex, therefore, is divided into primary zones and zones of evolution. Every psychic process, therefore, results from a successive integration of these elementary cortical areas. A complex mental process cannot make itself manifest without the integration of many or even all parts of the cortex and the absolutely necessary participation of the evolutive zones. One cannot talk of inhibitory centers, because inhibition is part of the function of the entire cortex and in fact of the entire central nervous system. By analogy one cannot talk of centers for the emotions as being separate from the areas in which intellectual functions are elaborated. These are always accompanied with varying degrees of emotional tone. Further, one cannot talk of a center for memories as distinct from the fields in which sensation and perceptions are elaborated, even granting that these two phenomena may make themselves manifest through the activity of different layers in the same area. Perceptions and memories which are habitually presented to our consciousness are already synthetic processes and not elementary sensations, therefore their localization is to be looked for in their elements, namely, the areas for sensations and their residual memories. Perceptions result from a fusion of sensations and memory images.

Just as the architectonic areas must be divided into primitive and evolutive, so must the elementary functions be distinguished as primitive or fundamental. The indispensable elementary functions are, for example, the visual images and the visual or light sensations with their residual memories. The evolutive sensations which are complimentary to these are, for example, the visual images with their associated motor components.

Our perceptions as related memory images are not a unit but an integration of elementary functions which cerebral activity produces by means of its sublimating processes. Thus the destruction of an elementary fundamental function renders useless the corresponding processes of perception, whereas the destruction of complimentary elementary functions may alter or suspend corresponding primitive elementary perceptive processes. These may not be completely abolished, however, because there is always the possibility of at least a partial reconstruction.

Every psychic function must be considered as a reflex resulting from the simultaneous synergy of various cortical areas, some with an afferent phase, some intermediary and one efferent, all of varying complexity. There is no physiologic or anatomic basis for the concept that the more highly evolved functions reside in certain centers. Ideational centers, distinct from areas in which the elementary functions are localized, do not exist. There also do not exist centers of association, intelligence, emotion, memory, will, consciousness. These have no other bases than our sensations, their respective residual memories and the association between them. One can understand how a small lesion may alter general functions, because this may cause interference with elementary functions resulting in the phenomena of diaschisis and thus interfering with movement, inhibition and automatism. In the cortical areas, as in other parts of the nervous system, the structures of movement are found ventrally and in man anteriorly to those of sensation. This holds true in spite of two apparent contradictions. If the visual or auditory areas are excited, movements of the eyes and ears, respectively, may result, but these are caused by the production of subjective images and are not to be considered as purely motor phenomena.

Not all psychic reflex arcs result in an efferent motor phase. Some cause the processes of attention and inhibition, and others simply modify our emotional state or consciousness itself. But all these psychic phenomena are of a reflex character which may call into play various elementary functions in many portions of the cortex. This is made possible by many intracortical and subcortical connections.

Those cortical areas that are concerned more directly with language naturally are the evolutive areas, but they in turn are to be further differentiated by the periods of their development both philogenetic and ontogenetic. The original of these areas is the auditory area, which with the zone of Wernicke in the posterior portion of the first temporal lobe form the forty-first and forty-second fields of Brodmann. The secondary zone is the verbomotor in the foot of the third frontal, which is Brodmann's field, No. 44. The third field in point of development of language is the verbovisual at the angular gyrus (Brodmann's area No. 39). There is some doubt as to the existence of a graphic area which directs the movements of the hand in the act of writing. This area is said to be located at the foot of the second frontal, but a study of the cito-architecture of this area does not show anything characteristic. Aside from these important primary areas, there are subsidiary areas which were studied above all by Henschen. There are the musical-sensory (vocal) and motor (instrumental), the area for the recall of number images and for designs and colors. It must be recalled, besides, that language is nothing but a means of expression, and therefore is closely allied to other modes of expression, such as mimicry, gestures, the assumption of attitudes, etc. These functions are represented in cortical fields, which one might call parallel areas. In these, the elementary functions of expression other than language are localized and can be externalized by the psychic reflex processes mentioned.

Language, however, cannot be an isolated cortical function. The primary verbo-auditory area, for instance, cannot be isolated artificially from the various sensory and motor areas with which it functions synergically. Neither can the secondary verbomotor area act alone without the assistance of the entire motor area of the cortex (ascending frontal, fourth field of Brodmann), especially its lowest portion. These areas necessary to the functioning of the special areas for language might be called associated areas. Finally, the function of language depends also on the integrity of extracortical mechanisms, such as the internal capsule, the subcortical ganglia and the brain stem. At once we see how inadequate the conception is of single or even circumscribed centers for language. In certain moments, the function of language may be determined only by the activity of the entire brain. The data supplied by study of the architectonics of the brain bear this out. Von Monakow showed that aphasia may be explained only on the basis of these studies. He was the first to insist that the conception of cortical centers containing representations of words was a false one. Verbal images are complex formations, and in the single cortical areas nothing is possible except elementary functions, and only by a complex synthesis of these functions is a verbal representation possible.

The vicarious assumption of the functions of one cortical area by another is said by some to be impossible. Each of the elementary cortical areas, in order to function, must have certain biologic characters—age, capacity for development, absolute integrity and certain anatomic connections with other areas. While this appears quite definitely true, it is premature in the light of our present knowledge to say that under certain favorable circumstances certain of the functions of one of these areas may not be assumed by another. Also it is impossible to deny that even though one of these centers may be more or less permanently damaged, its functions may be regained with defect or entirely. Nevertheless, it is quite striking that 90 per cent. of patients with motor aphasia do not get well.

Interruption of association pathways also cause aphasia. The Vogts have shown that in Brodmann's field 6α the connections between the large pyramidal cells are intracortical, whereas in the postcentral region the pathway is subcortical via the U bundle. Here an interruption of the subcortical association pathway would destroy function just as seriously as cortical injury. Recalling that all psychic functions should be considered as reflex phenomena, interruption of the reflex arc in any part of its path causes abolition of function. These reflex psychic processes as they apply to language are not as equally developed in both hemispheres as are the purely motor and sensory functions, which are exactly paired. This is probably due to an economy in the elaboration of already complex synthetic processes, so that they may be almost exclusively confined to one hemisphere—the left. The psychic reflex for the function of language comprises an afferent phase consisting of sensations and perceptions, the recalling of images, the reintegration of words as sensory images, the reinvocation of motor word images, and finally the impulse to express these by various motor efferent pathways. These processes all take place chiefly in an area of the cortex supplied by the middle cerebral artery, and it is conceivable that changes in the arterial irrigation of one area may result in a deficiency of function which is felt in the entire cortical area for language.

The primary area for aphasia (zone of Wernicke, Area 41 of Brodmann, including anterior portion of internal transverse gyrus and Brodmann's Area 42 including the posterior portion of the external transverse temporal gyri). This area also corresponds fairly well to the first transverse temporal gyrus of

Hesch. These areas have a characteristic myelo-architectonic structure very rich in multistriate bundles, with a definite line of Kaes-Bechterew. Here are stored the auditory sensory elements of speech; not auditory word images held in a receptacle as any object might be in a box, but held in a formative stage which, when acted on synergically by the activity of other cortical fields, results in the transformation of sensation into perceptions, and finally by association into symbolic expression and the spoken or written word. Until symbols are formed in this way and are recalled and recognized by the speaker, speech is not a complete act. For example, he who has learned a strange language without hearing it spoken cannot understand that language well when it is first heard.

If it is true, as Wernicke states, that the verbo-auditory zone corresponds with the auditory projection tonal area from si 1° to sol 2°, the method of development from a primitive sensory area to a primary evolutive (speech) zone is easily understood. This primary zone has intimate connection with the insula (island of Reil) and with Broca's area, by means of the uncinate bundle, with the supramarginal area, with the striate and with the thalamus by means of the inferior longitudinal bundle, and with the opposite supramarginal and visual areas, and these areas on the same side probably via the tapetum. The verbo-auditory zone precedes the development of the others. The child first hears the mother's voice, then imitates it later by articulate speech. [Is this all the process? Is not language first of all an instinctive thing—a vital need to commune with one's fellows? Does not the child really make its first sounds instinctively? It cries because of pain, hunger, discomfort, etc. Then it finds the production of sound pleasing and the movements of tongue and lips give pleasant proprioceptive sensations which lead it to repeat sounds, at first, gutterals and labials perhaps, then others. Eventually the mother is heard to repeat sounds which mean something to the child as it sees and feels and tastes the thing that the mother is making sounds about. Thus it gets symbols, perhaps chiefly but certainly not exclusively by way of auditory sensory paths.] (Osnato, M.: The Integration of Speech and Intelligence, Neurol. Bull. **3**, No. 2 [Feb.] 1921.) If anything interferes with the function or development of the auditory sensory perceptive area, naturally the evolutive auditory speech area becomes inactive or disturbed even to the point of abolition. So it is with any primitive sensory function. Its disturbance always is reflected in the serious impairment of the corresponding evolutive area with which it is connected. The converse of this law is not true to anywhere near the same degree. Injury to the primary verbo-auditory area does not necessarily affect hearing.

Lesions of the verbo-auditory area cause so-called sensory aphasia, difficulties of understanding, word deafness, paraphasia, poor repetition of the spoken word, disturbance of internal speech, conservation of spontaneous speech with many mistakes, word blindness, alexia, printing only to copy, and other forms of agraphia. There is disturbance, therefore, of all forms of language expression. Language is not entirely abolished because the other (secondary tertiary, subsidiary, etc.) areas concerned with speech are not disturbed. The only specific loss is the word deafness and the disturbance of internal speech. The other symptoms are due to disturbance of the other centers which have depended for their existence and development on the development of this primary center. They, nevertheless, especially the secondary (verbomotor) center, have a certain independence as the result of their long philogenetic and ontogenetic history and continue to function as best they can without the complete aid of the disturbed primary center. These same disturbances occur if the cortex is spared and the association pathways to and from the verbo-auditory zones

are injured. It is conceivable that injuries to the tertiary and lower subsidiary centers may have the effect of disturbing the functions of this primary speech center with the same sort of speech defects mentioned above discoverable on careful examination. As clinical evidence of this, witness the denial of many that any case of pure word deafness has ever been reported, and word deafness alone should result if the primary speech center were an independent circumscribed station for storing auditory word images.

The secondary area of aphasia is the verbomotor foot of the third left frontal and Broca's area including Field 44 of Broadmann and the operculum of the island of Reil. The work of Nissi, Knauer, and Catalano shows a characteristic architectonic structure. In the deepest pyramidal layers, there are scattered irregularly or in groups large pyramidal cells much larger than those of the third layer and of the ganglion elements. These resemble the anterior horn cells of the cord. The internal granular layer is so sparse in comparison with sensory areas elsewhere in the cortex that Nissl says it does not exist. The cells are arranged in columns rather than tangentially. The cortex of this area is large, and the pyramidal cells of the third layer are not much like those of the second layer so that there is no real pyramidal stratification. The white bundles are less abundant than in the precentral convolution. The characteristic myelo-architectonic feature is the absence of the striae of Kaes-Bechterew. The posterior confines of this area may not correspond with the precentral sulcus and may not be limited exactly to the foot of the third frontal. The area has connections with the second frontal, with the verbo-auditory area by way of the uncinate bundle through the insula, with the visual area and the supramarginal gyrus through the arcuate bundle and with the precentral area. There are also commissural connections with the same area on the other side. The desire of other authors to extend this area beyond the foot of the third frontal, where Brodmann's Zone 44 is limited fairly definitely, is due to the fact that motor aphasia may be caused by lesions in the pathways connecting it with the second frontal, the island of Reil (Déjerine) the operculum (von Monakow) etc.

Wernicke does not believe that Broca's area has a purely motor function, but it has something to do with the elaboration of tactile images connected with speech and the proprioceptive sensations issuing from articulated speech. It also serves according to Wernicke as a point of departure for stimuli to the nearby motor areas controlling the muscles of expression. Nissl has expressed the idea that Broca's area is an area for words of little importance ("belanglos") or infrequent use. While this may appear bizzare, it may be possible that in this evolutive center sensations and their memories connected with words infrequently used are integrated, while automatically used words are stored in the motor centers of the precentral area. This may explain why in some motor aphasic polyglots, the mother tongue is least of all disturbed and is the first to be restored if function returns. When this happens the most usual phrases and words are regained, and finally if recovery takes place the last acquired languages are respoken.

The symptoms of motor aphasia are aphemia, impossibility of repetition after dictation, loss of internal language, inability to read aloud, mild visual psychic and auditory psychic disturbances and agraphia. The specific lesion is aphemia, the other symptoms are the expression of secondary disturbances in the primary, tertiary, subsidiary and other centers. Lesions of Broca's area, while grave prognostically, are not as bad as lesions of the primary area which is so fundamentally important for speech. The recovery of patients with motor

aphasia is explained by vicarious function of Broca's area of the opposite side, recovery from a limited or temporary lesion, or by the preservation of automatic speech in the ascending frontal convolution. It is interesting to note that recovery of the power of expression takes place in an inverse manner from normal learning. It is not the syllable or letter that is first spoken, but whole words or phrases. This is probably because long practice has made the reading of words automatic, not as in childhood a matter of reading letters, then syllables, then words. In these cases, often reading is preserved especially in well-educated and well-read persons, making the matter of recovery so much easier. Conversely, in the ignorant, who must depend so much on the verbomotor and verbo-auditory images for reading, motor aphasia produces a profound reading defect which is not recovered easily.

There are few cases of motor aphasia on record in which there were no agraphic disturbances, their seriousness depending on the degree of interruption of the connections of the graphic with the verbo-auditory and visual centers. Writing from dictation is also impossible because of the loss of internal speech.

The many cases of injury to Broca's area without aphasia and motor aphasia without lesions in this area should have all been checked up by histologic and architectonic studies. Only when it shall be proved that Brodmann's Area 44 (which often does not correspond with the foot of F_3) was not injured, can the claims of Marie and others be considered valid. Besides, there is always the possibility of a vicariously functioning right F_3 or of the preservation of automatic speech in the precentral lobule.

Tertiary Speech Area: This is the verbovisual area, left gyrus angularis, field of Broadmann 39 connected with the visual area of the same side, with the auditory speech area by way of the inferior longitudinal bundle, with the angular bundle and supramarginal gyri and the auditory speech area of the opposite side via the tapetum, with Broca's area and F_2 through the uncinate bundle. The angular gyrus has connections with the optic radiations of Gratiolet.

In the function of reading, there is a continuous association between visual images of letters and syllables and the corresponding verbo-auditory and verbomotor images. While for some persons, visual word images depend on the representation visually of single letters, for most people the more common words and expressions are not differentiated but recognized at a glance. This is done by the recognition of the proper grouping of the single letters, probably due to modern methods of teaching in our schools. The entire word is recalled by visual word or auditory word images and not usually by the single letters. While at first words are read by articulating single syllables, as reading exercises make us more proficient the visual word areas assume a certain independence from the verbomotor area, and entire words are recognized and read without syllable uttering. The perfect functioning of the verbovisual area, however, presupposes the integrity of the verbo-auditory and verbomotor areas and of the connections between them. The degree of defect resulting from injury to this area depends, nevertheless, on the amount of autonomy developed by each of the other areas and the extent of the lesion.

The unilateral situation of the visual word area is not certain. Visual images of the right hemisphere are connected with the visual word area of the left hemisphere, not as Wernicke says by an extension of the visual pathways beyond the actual projection areas, but by means of association and commissural pathways between the two hemispheres. Wernicke believes that visual word images are not stored exclusively on the left side of the brain, because in cases of right hemianopsia word blindness does not exist generally. According to von

Monakow, word blindness is due to lesions of the angular gyrus which interrupt the subcortical pathways which lead to the visual word center of the left side and the transcortical pathway which connects the visual word area of the right side with the area for auditory-word images. This neither of the visual word areas could function.

The symptoms of injury to the visual word area are word blindness or alexia occasionally only for letters with or without hemianopsia, depending on whether the lesion is cortical or not. If the lesion is subcortical, one may have hemianopsia—complete or quadrantic. A purely occipital lesion does not cause alexia, providing the temporo-parietal area is not involved. In these cases of pure occipital lobe lesions, visual images are easily recalled, and reading is not affected.

In cases of word blindness, copying is possible, or spontaneous writing is preserved without, however, the ability to understand what has been read or to write from dictation. These patients may not be able to name objects without feeling, tasting and smelling them. Psychic blindness cannot exist without word blindness, but a smaller lesion may result in pure word blindness alone. This is probably because the latter function has been more recently acquired phylogenetically and ontogenetically.

The existence of an area for graphic images is not admitted by all. It is localized in the left second frontal. This area does not have a characteristic cyto-architectonic structure differing from Brodmann's area No. 6 which is more extensive, nor does it differ from areas further forward. Wernicke, Kussmaul, Gowers, von Monakow, Bianchi and Déjerine deny that it exists, particularly because it is possible to write with any movable part of the body. This does not exclude, however, the possibility of the existence of an area in the motor cortex which has the function of fixing the various elements of movement necessary to direct the hand or any other part of the body during the act of writing. In this center, tactile, muscle sensibility and proprioceptive sensations may be integrated to the point of developing a rapid automatic act such as writing. In simple cases of agraphia, it is conceivable that the aphasia may be mild or even absent (as in a case of Gordinier), and on the contrary, it is possible to have cortical motor aphasia without agraphia. Generally in motor aphasia, copying is possible, which shows that there is preservation of the sense of direction of movement. Wernicke's idea that the graphic center corresponds with the area for fine movements of the right hand will be tenable only when it is demonstrated that paralysis of this area determines a loss of ability to write with the left hand. Marie objects that writing dates from a too recent period of the development of speech in man for the localization in the cortex of an area which governs this function. Nevertheless, this act must be made possible only through the interrelation of certain definite areas of the brain. Déjerine objects that graphic images cannot exist, because the patient who cannot write spontaneously often copies very well. However, spontaneous writing is a much higher intellectual act than copying and must require the integration of many more cortical areas than simple copying. Déjerine further objects that in typewriting the element of direction of movement does not enter. In cases of agraphia, in which there is even an impossibility to form words with blocks or to type words, one must of necessity believe that internal speech is so badly injured that the lesion is profound and extensive and involves much more than any single area of the brain.

Because of the close connection between the motor word area and the graphic area and in turn of the first with the auditory-word area, lesions of the former with the latter determine agraphia and paragraphia. Unilateral lesions of the arcuate association bundle may give unilateral agrapha as in the case of Pitrés, in which after recovery from a right hemiplegia agraphia continued limited only to this side, while the patient could write with the other hand.

The subsidiary speech centers were studied chiefly by Henschen: auditory centers for numbers, vocal music, instrumental music; motor centers for numbers and vocal or instrumental music; visual centers for numbers and vocal music and for designs, also possibly motor and visual centers for colors.

When one talks of a music center it is not intended to convey the idea that musical notes are stored there, but that therein is localized an elementary function which by synergic action with other areas may serve to recall musical notes or symbols. This center for musical vocalization is localized by Henschen in the inferior margin of the pars triangularis of the third left temporal. A center for the understanding or appreciation of music is situated in the left temporal pole and another for musical notes at the junction of the second parietal with the angular gyrus.

Instrumental amusia has been localized with difficulty, because musical instruments may be played with the hands, mouth, etc. Musicians have lost the power to play the violin and not the piano. Bianchi has always maintained that the right hemisphere has great importance in connection with singing, particularly of popular songs. He says that this type of singing may be preserved if the right hemisphere is unharmed.

Parallel speech centers are closely connected with the higher speech areas. These parallel centers control general modes of expression, such as mimicry, gestures, attitudes, the movements of the eyes and eupraxia. These functions depend on the prefrontal and parietal lobes and on the insula. Lesions of the prefrontal areas other than the second and third frontal cause stammering, scanning and dysarthria. The insula is closely connected with the lenticular nucleus. Its architectonic structure in man is similar to that of animals, and it is reasonable to assume that it controls emotional expression. Lesions of the insula may cause an aphasia of conduction by compromising the uncinate bundle which connects the auditory center with the motor centers. The insula is also connected closely with expression by mimicry, and determines an adequate emotional response when the spoken word is uttered. Primitive emotional speech may also depend on insular function.

The reflex psychic arc for language, therefore, passes through these stations, acting synergically with other secondary, tertiary, subsidiary and parallel centers by means of association tracts, extracortical areas, projection systems, sensory systems, the general auditory sensory area, the auditory word area, the Broca area, the motor area, the insula, and so on. In a complex speech integration, probably the entire brain functions.

Association Areas and Pathways: The auditory speech area cannot be artificially isolated from the general auditory areas, neither can the visual psychic from the occipital striate areas, nor can either of these centers be separated absolutely from olfactory or taste areas. The motor speech area likewise cannot function independently of the general motor area. Indeed, some believe that automatic speech is localized in the general motor areas. Interruption of the arc in the lower part of the ascending frontal abolishes the efferent arm of the

arc, causing anarthria or a residual dysarthria. Speech may be even more seriously disturbed if the lesion in the ascending frontal is deep, involving the centrum ovale. Alexia, agraphia, disorders of understanding, etc., may occur.

Extracortical Apparatus: In these are included the projection systems of sensation and movement and the internal capsule, lenticular nucleus, etc. Mingazzini and P. Marie have insisted that motor speech functions of a certain type are carried in the connection between Broca's area and the anterior area of the left putamen, and the second relay of these fibers commences at this point. There are connections here with fibers from the opposite Broca area and from the ascending frontal. Mingazzini believes that aphasia or anarthria may occur with lesions in this part of the putamen. The work of Vogt seems to invalidate the claims of Marie and Mingazzini. From the point of view of phylogeny, it seems impossible to believe that the lenticular nucleus has any important speech function. Déjerine and von Monakow point out that anarthria occurring in lenticular lesions is due to lesions of the internal capsule. The fibers in the capsule from the ascending frontal and the foot of the third frontal are in immediate contact with the putamen. The confusion following the teachings of Maries and Mingazzini (famous quadrilateral area of Marie) grew out of improper conceptions of the difference between anarthria and aphasia.

This discussion shows how vast may be the territory involved functionally in aphasia. In the great majority of cases of aphasia, the lesions have been irregularly distributed, multiple or diffuse, involving often even the meninges and entire cerebral vascular systems. Even when single lesions are found there are other general factors, such as paralysis and various motor phenomena, hypertension, etc. It is plain also that any lesion causing aphasia will interfere seriously with the rest of the complex psychic reflex arc concerned with the speech function. Besides there are individual variations in the exact location of the various so-called speech centers. Besides topographic studies, architectonic studies are imperative. The distinction between the various clinical types of aphasia is more or less artificial. This is especially true when one attempts to distinguish between cortical and subcortical types. There has never been reported a single well-studied case of aphasia in which the lesion was purely cortical. One doubts even the existence of cases of "pure" motor aphasia.

The aphasic mentality is not comparable to real dementia. As Bianchi has pointed out, the aphasic person can still think. While it is true that words fix the symbolic importance of every image, nevertheless, this process is secondary from an evolutionary standpoint to the development of thinking. The aphasic patient can still make use of the associations between nonverbal memory images. He can still make use of memories without expressing himself in terms of written or spoken symbols, provided, of course, the lesion has not been so severe that even these memories are destroyed.

In closing, the author calls attention to the fact that even Head has fallen into the error, made by so many, of neglecting the basic consideration of the problem of aphasia. Head has neglected to correlate properly his well studied clinical material with known anatomic and histologic facts. Head's classifications (verbal, syntactical, nominal and semantic aphasia) are artificial and based on presumed clinical differences. Only anatomic and even more important, cyto-architectonic studies will be of lasting value in this work.

OSNATO, New York.

COMPARATIVE INVESTIGATIONS OF INSECT BRAINS WITH PARTICULAR REFERENCE TO THE MUSHROOM STRUCTURES (CORPORA PEDUNCULATA DUJARDINI). RUDOLF BRUN, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow **13**:144-172, 1923.

The central nervous system of insects, as is well known, has a segmental arrangement and consists of a chain of paired ganglia which are united longitudinally and transversely by commissures. While most of these ganglia lie ventral to the intestinal tract, three anterior pairs are fused into a mass that lies posteriorly and forms the ganglion supra esophageum, cerebral ganglion, or brain. This ganglion is united by a pair of particularly massive connections with the second complex that lies under the intestinal tract, and is known as the ganglion mandibulare, maxillare, labiale or infraesophageum.

The superficial resemblance of the abdominal ganglion chain of arthropods to the sympathetic chain of vertebrates induced Cuvier to homologize the two. Johannes Müller subsequently discovered in insects a separate visceral nervous apparatus and refuted this homology as untenable. Numerous investigators have proved beyond doubt that the abdominal ganglion chain of insects corresponds to the spinal cord of vertebrates and the supraesophageal ganglion, to the brain. Brun shares the opinion of most writers, namely, that the infraesophageal ganglion is not to be considered a part of the brain but belongs to the abdominal chain ganglia. The chief differences between the nervous system of vertebrates and that of insects are the more marked bilateral segmental character and the absence of inversion of the medullary plate, the nervous system remaining in its morphogenesis, at the stage of the medullary plate. The ventral position of the ganglion chain is readily explained by the general inversion of the organism in a dorsoventral direction, so that the abdominal side of an insect corresponds to the dorsal side of a vertebrate.

The fusion of the three preoral head ganglia into the supraesophageal ganglion results in a three-lobed structure. The anterior lobe, or protocerebrum, consists chiefly of the primary optic centers of both complex and simple eyes, as well as the corpora pedunculata, or mushroom bodies of Dujardini. The second lobe, or deutocerebrum, is concerned with the sense of smell. The third lobe, or tritocerebrum, is very small in most insects, and probably gives rise to the N. labialis.

Dujardini was the first to discover the large, dorsally situated and lobulated structures in the brain of the social hymenoptera. These structures consist of two peculiar goblet-formed bodies with a large stem that proceeds to the interior of the brain, and for this reason he called these bodies the *Corps Pédonculé*. They are more highly developed among the insects. The bodies have on their exterior a structure resembling the marginal gray matter, and would resemble the cerebellum if the granular and the molecular layers were inverted. In the annelides and in the lower forms of crabs, no bodies corresponding to the corpora pedunculata can be found. This is also true of spiders. In the centipedes, this structure can be recognized. As stated before, the fullest development occurs in the social hymenoptera, the bees, wasps and ants. These bodies may form the anatomic substratum of the intellectual activities of these remarkable insects. Forel discovered that the pedunculate bodies of ants vary in size; those of the workers are largest, of the female smaller, and of the drone rudimentary. Brun studied the comparative size of various parts of the brains of a series of insects. He made the interesting observation

that among the ants, *Formica fusca,* which represents a phylogenetically older type, out of which such types as *Formica rufa* developed in the late tertiary period, exhibits the more primitive instincts, less psychic plasticity, better ability of orientation by the sense of smell, and a greater dependability on the objective senses. In *Lasius umbratus* the size of the corresponding part of the brain is only a trifle over one-fourth of that of *Formica rufa,* so that this group would represent microcephalic individuals among the ant population, and the male would have good reason for being the most ignorant member among the ants.

The lateral corpus peduncularis is larger than the medial, and phylogenetically probably represents a later acquisition. In the case of the orthoptera (*Locusta viridissima*) it is found that the brain of this large insect, which measures 3.1 cm., is only 3.8 times as large as that of the red ant. Proportionately, the brain of the grasshopper is one-third as large as that of the red ant, and the relative size of the corpora pedunculata of the grasshopper is barely one-fifth that of the ant. In the case of the lepidoptera, the moths and the butterflies, *Macroglossa stellatorum* is three times as long as the wood ant and about twenty times larger in volume, yet the corpora pedunculata are actually smaller than those of the ant, the maximal cross section being only about 4.3 per cent. of that of the entire brain, and thus about one-seventh as large as the corpora pedunculata of the ant. Even when the large lobus opticus of the butterfly is disregarded, these bodies represent only 16 per cent. of brain volume in comparison to 41 per cent. of brain volume in the ants. In the diptera, flies and moths (*Calliphora vomitoria*), the corpora pedunculata are decidedly rudimentary and of the globulus type, which is the least highly developed form. Here the cross section of the corpora pedunculata is 0.22 as large as that of the ant, the body of which is only half as long. In general among the insects, it is found that the poor development of the corpora pedunculata is compensated for by a correspondingly greater development of the visual centers.

Although we are pretty well acquainted with the finer histologic structure of the corpora pedunculata, little is known concerning the central fiber connections. The elements making up the granular portion are of uniform size in the lower insect types, such as the grasshopper, the fly and the butterfly. On the other hand, in the ants, the cells in the center of the calices are distinctly larger and paler than those at the periphery. As the dorsal bundle passes dorsad, it decreases in volume gradually and disappears basal to Viallanes' bridge, in a network of fibers that spring from the giant cells of the intercerebral region and pass to the infraesophageal ganglion.

The view of most authors that the axons leave the corpora pedunculata to pass to motor nuclei of the infraesophageal ganglion, thus forming a kind of pyramidal system, must be denied. Brun believes that the cells of the corpora pedunculata are mnemic in function, and that the fiber tracts that pass into the pedunculi must be regarded as association elements which connect the mnemic activities with the motor apparatus. The cells corresponding to the pyramidal system are probably the large intercerebral, somatrochrome elements. The dorsal bundle of the corpora pedunculata probably comes in contact with these cells through the medium of spindle cells. The pyradimal fibers seem to pass into the infraesophageal ganglion and the abdominal ganglion chain. The exact pathway is not known. The complicated instinctive and

automatic activities, fixed by heredity, are a function of the corpora pedunculata only indirectly so far as these bring this "subcortical apparatus," which presides over these instincts, under the control of the senses.

WOLTMAN, Rochester, Minn.

BRAIN ABSCESS. HANS CURSHMANN, München. med. Wchnschr. **70**:1258-1259 (Oct. 5) 1923.

The subject of brain abscess is of special importance because of the need of early recognition and the usual good results following operation. Otitic abscesses are probably the most common, while traumatic abscesses are less frequent. This was especially true during the war period. Following trauma, a brain abscess may develop immediately, running more or less the course of a stormy meningitis with few associated focal signs. There may be a latent period, however, and only later localizing signs may develop. As a rule, traumatic abscesses are single, and are situated on the side or near the region of the injury.

Occasionally brain abscesses follow furuncles or phlegmons of the scalp, sinus infections, or other localized processes. In this group, the abscess usually results from direct extension. It may, however, be metastatic. Metastatic abscesses are especially common in empyema and other lung infections. Metastatic abscesses in endocarditis are surprisingly rare. In a review of 400 cases of endocarditis, Stahl was unable to find evidence of a single brain abscess. Idiopathic brain abscesses are mentioned only because the cause of infection is unknown.

Anatomically one may have localized encapsulated abscesses or diffuse abscesses, either single or multiple. In the early traumatic abscesses, there are signs of severe meningitis with little evidence of localizing value. In the late traumatic abscesses, as also in the metastatic, there is usually a latent period during which time there may be an occasional headache (temperature, if present, is only slight), a loss of weight, apathy or "pseudoneurasthenia." Bradycardia may be present, while choked disk is usually absent. The latent period may extend over months or even a year. If the abscess is large and situated in focal areas, localizing signs may develop. Abscesses, especially of the right frontal and right temporal lobe, may run a symptomless course. Headaches, if present, may be variable in character. Localized tenderness is often present. In the period of activity, vomiting, apathy, or deep coma may develop. Even during this period, temperature, choked disk and leukocytosis may be absent. Lumbar puncture may show an increase of pressure, with excess of globulin and some cells; but frequently the fluid is normal. It is well to bear in mind the great danger in posterior fossa abscesses, the same precautions being necessary as in tumors of this area. While the Neisser brain puncture is condemned by many, it should not be overlooked, especially if operative procedures are at once instituted in case pus is discovered. Roentgen-ray diagnosis seems to be of little avail, while pneumo-encephalography is confronted with even more danger than in brain tumors. If meningitis develops, operative procedure seems inadvisable. Occasionally there may be a spontaneous degeneration of an abscess.

The otitic brain abscess is the most common type, probably making up about one third of all brain abscesses. It is more apt to follow a chronic than an acute middle ear process. Extension to the base of the temporal lobe

or to the anterior portion of the cerebellum is most common. As in any other abscess, there may be three stages: the initial stage of abscess formation, the latent period and the stage of activity.

Otologists state that in right temporal lobe abscesses, hearing disturbances of the opposite ear are to be looked for because of the crossed hearing fibers. Left temporal lobe abscesses may lead to sensory aphasia, optic agnosia, difficulty in reading, mild paraphasia, and even hemianopsia, hemiparesis and involvement of the oculomotor nerve, causing pupillary changes or ptosis if the abscess is large. In abscesses of the cerebellum, dizziness, falling to the side of the lesion, diminished corneal reflexes on that side, hypotonia, past-pointing and nystagmus to the affected side may be present. Choked disks are quite frequent in cerebellar abscesses. If the abscess is large, the cranial nerves and pyramidal tracts may be involved.

The diagnosis of an otitic brain abscess is usually difficult. This is especially true in children or in apathetic patients. One must differentiate the process from otitic meningitis, extracerebellar subdural abscess, pachymeningitis or sinus thrombosis. Operative results in otitic brain abscesses are favorable as a rule, the figures varying from 15 to 53 per cent. of recovery.

In other forms of brain abscess, the diagnosis may be equally difficult. In all skull injuries, in chronic pulmonary and pleural affections, this possibility must be kept in mind and the presence of a latent period must not be overlooked. In any brain abscess, an attempt should be made to institute surgical intereference before cachexia sets in. This is especially advisable in view of the fact that the results of operative procedure are so encouraging.

MOERSCH, Rochester, Minn.

EXPERIMENTAL ANATOMIC INVESTIGATIONS CONCERNING THE PROJECTION OF THE RETINA ON THE PRIMARY OPTIC CENTERS. DR. B. BROUWER, W. P. C. ZEEMAN and A. W. MULOCK HOUWER, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow **13**:118-137, 1923.

Up to the present time there has been no experimental investigation of the projection of various parts of the retina on the corpus geniculatum externum and the corpus quadrigeminum anticum. Pick and Herrenheiser followed the secondary degeneration resulting from lesions of the retina in rabbits and other mammals, in the optic nerve, the chiasm, and the optic tract, but did not study the primary centers in the brain. This also applies to the work of Usher and Dean. Recently Lubsen studied the degeneration in the tectum opticum in fish (*Leusiscus rutilus*) by the Marchi method. Zeeman found that certain portions of the retina and optic nerve, when injured, result in a well localized degeneration in the tectum opticum; fibers which arise caudal to the papilla end in the anterior portion of the tectum, and vice versa. Lubsen found that the upper retinal fibers reached the lower portion of the tectum opticum, and vice versa. Pick and Herrenheiser produced partial destruction of the retina of rabbits by means of the galvanocautery and by a hook. The fibers were found to retain their relative positions throughout their course in the optic nerve and tract. The crossing in the chiasm occurs in an orderly manner; the fibers cross horizontally but not vertically.

In the following experimental work, Zeeman and Houwer produced lesions in the retina of rabbits and cats. In operating on the rabbit, atropin and cocain were instilled into the eye. Following luxation of the eye to the

outside, a long sharp needle was passed through the bulbar conjunctiva and the sclera on the temporal side, a few millimeters anterior to the equator under the external rectus muscle into the vitreus toward the optic nerve. The point was guided by observing it by means of a head light and a plus 4 diopters lens before the eye and a plus 20 in the ophthalmoscope. The desired portion of the retina was thus injured. For the temporal quadrants the needle was introduced from the nasal side. Ophthalmoscopic examinations were made daily, and an accurate sketch of the lesion was made before killing the animal. The same method was followed in cats, however, without luxating the eyeball and under ether narcosis. The left eye was always used, and the animal was killed after eighteen days. Marchi sections were made of the optic nerve, the chiasm, the optic tract and the primary optic centers.

In a rabbit in which the left eye was removed, only fibers on the opposite side were seen to be degenerated. In three rabbits, the upper retinal quadrants were injured, in one the upper temporal quadrant, in a fifth the upper nasal quadrant, in a sixth both lower quadrants, in a seventh both lower quadrants and the upper temporal quadrant, in the eighth and ninth the lower nasal quadrant, in the tenth the lower temporal quadrant; in the eleventh, a horizontal section was made immediately above and below the papilla, and in the twelfth multiple lesions of the retina were produced. Four cats were experimented on; in two of them the upper quadrant was destroyed, in the third the nasal half, and in the fourth the upper temporal part of the retina.

The following conclusions were drawn: Even in the optic nerve there is no absolute representation of the various portions of the retina. Fibers from the upper portion of the retina tend to lie above, those from the lower half, below; the upper tend to lie laterad, the lower mesad. They tend to intermingle. The same is true of the nasal quadrants and of the temporal quadrants. In the chiasm, there appears to be no rule of localization and, all the fibers being intermingle. In the optic tract, the upper retinal quadrants are represented chiefly in the ventromedial portion of the tract, and the lower quadrants of the retina in the dorsolateral part of the tract. This rearrangement takes place principally in the chiasm, so that it may be said that in the chiasm of the rabbit there is crossing both horizontally and vertically. Even in the optic tract, there is some intermingling of the fibers.

As far as the external geniculate body is concerned, Henschen is correct in his statement that certain portions of the retina are limited to definite portions of the geniculate body. The upper half of the retina always lies ventral to the lower; fibers from the temporal half occupy a medial position in the external geniculate body, whereas the nasal fibers are represented laterally. The lines of division in the external geniculate body are not exactly horizontal and vertical. The nasal quadrants tend to lie under the temporal. In the cat, this reversal seems to be a little more complete than in the rabbit, and is probably related to the different position of the eyes in the head. Physiologically speaking, the picture of the outer world which appears inverted on the retina again becomes upright in the first optic stations of the brain. In regard to the question of overlap, it may be said that the projection areas of the four quadrants are sharply delineated. In general, the doctrine of neurobiotaxis of Ariens Kappers seems to apply, as quadrants which are frequently stimulated by the same impulses, for example the two inferior quadrants, are in contact over a larger surface than others, as the inferior temporal and the superior nasal, which are seldom stimulated simultaneously. The smaller ventral, external geniculate body shows a fine granulation indicating that

some of the optic fibers end in it. Most of the fibers seem to pass by this nucleus. The nucleus is not related to the cortex but is probably related to the anterior corpus quadrigeminum.

Although it has for a long time been assumed that the pulvinar has primary optic fibers going to it in the rabbit at least, the fibers seem to pass by it, bending around it in order to reach the anterior corpus quadrigeminum. The pulvinar of rabbits may not be homologized with that of the higher mammals without qualifications. In the cat still fewer fibers are seen to end in the optic thalamus. Minkowski was unable to demonstrate any secondary changes in the pulvinar of apes and men after destruction of an eye. The pulvinar radiations become progressively smaller as one ascends the animal series, which one would not expect to be the case if it were related to the primary visual fibers, as the pulvinar becomes progressively larger. This would also explain certain clinical facts better. Lesions in the external geniculate body, with preservation of the pulvinar, result in a total hemianopsia; however, lesions in the pulvinar without involvement of the external geniculate body are unassociated with any disturbance in the field of vision. Brouwer states that these findings must be checked by the method of secondary cell atrophy and that the conclusions drawn may not as yet be regarded as applying to the higher mammals.

WOLTMAN, Rochester, Minn.

THE COG-WHEEL PHENOMENON AND ANTAGONISTIC INNERVATION. ROBERT BING, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow **13**:77-87, 1923.

Statistics prepared by Bing and Staehelin in 1922 reveal that about one-half of the patients who survive the primary illness of encephalitis sooner or later develop the parkinsonian syndrome. In 1901, Camillo Negro described a characteristic phenomenon seen in paralysis agitans but also noted in the parkinsonian syndrome of encephalitis which can be demonstrated by pronating and supinating the forearm or flexing and extending the elbow, at which time it will be found that the movement is interrupted by rhythmically appearing resistances, as though the elbow joint consisted of a cog-wheel mechanism. Negro described this as the "fenomeno della troclea dentata." As has been shown since, it is not limited to the elbow musculature but seems to be common to all striated muscle. The name cog-wheel phenomenon was suggested by Mayer.

This phenomenon is more common and more marked in passive than in active movements. When noted in the extra-ocular movements it may suggest a nystagmus. This pseudonystagmus is clearly not a cerebellovestibular syndrome, but is of mesencephalic origin. When the two opposing muscles involved in the movement, for example the triceps and biceps muscles, are palpated simultaneously, the trepidation will be felt mostly or entirely in the elongating muscle and not in the contracting muscle. This is noted on active as well as on passive movement. This observation is not in accordance with those of certain other neurologists who believe that the phenomenon originates in the contracting muscles and is dependent on an insufficient dosage of the contracting impulse, requiring frequent reinforcement.

Negro believes that it is an expression of sarcoplasmic rigidity, a view not accepted by Bing. According to Nóvoa Santos, the syndrome results from disturbances of the paleokinetic system and is analogous to the ankle clonus seen in lesions of the neokinetic system. The rigidity of the parkinsonian syndrome is dependent on a disorder of the striate body and of the hypothalamus in

somewhat the same manner that increased tonus occurs from lesions of the pyramidal system; the phenomenon therefore represents a clonus of the parasympathetic portion of the muscle. Bing objects to this explanation, since Müller has shown that there is no certain proof of the innervation of striated muscle by the vegetative nervous system, and since no disturbance of tone could be demonstrated by Hess after removal of the abdominal sympathetic system. According to the author, the cog-wheel phenomenon is dependent on the abolition of inhibitory impulses arising in the globus pallidus and associated structures, passing via the ansa lenticularis to the nucleus ruber, resulting in an increase in the cerebellorubral tonus, just as the tonus dependent on anterior horn cells in the spinal cord increases after the cortical inhibition has been removed. In the first case we get the extrapyramidal rigidity of Parkinson's disease and the parkinsonian syndrome of encephalitis, and in the second case the well known spasticity of pyramidal lesions. Tinctura veratri viridis is supposed to have some influence in reducing this tonus.

HENRY W. WOLTMAN, Rochester, Minn.

THE FUTURE OF MENTAL DISEASE FROM A STATISTICAL VIEWPOINT. HORATIO M. POLLOCK, Am. J. Psychiat. **3**:423-434 (Jan.) 1924.

Pollock sets forth some interesting statistics concerning mental disease. In the year 1880, 40,942 patients were under treatment in public institutions in the United States; in 1920, there were 232,680—a six-fold increase. According to the ratio per 100,000 of population, there were 81.6 in 1880 as against 220.1 in 1920. The inference is not that there has been such a tremendous increase in mental disease, but that the public has more confidence in institutions and, furthermore, the institutional death rate is less than when the patients are treated at home. It is somewhat paradoxical, but true, that mental disease increases as physical disease decreases. For instance, if an epidemic kills a relatively large proportion of the population in the second decade of life, it necessarily removes many persons who would have developed insanity had they lived long enough. On the other hand, if, as is actually the case, the span of life has been lengthened by from eight to ten years, then it is obvious that certain types of mental disease which are peculiar to old age, such as senile or arteriosclerotic dementia, will have a large incidence. A second statistical principle which Pollock clearly proves is that the rate of mental disease is higher in cities than in rural districts. On the basis of first admissions to the New York State hospitals, the average annual urban rate per 100,000 is 69.5; the rural, 42.5. That this will become a serious factor in the incidence of mental disease is clearly apparent from the fact that, while in 1880 only 28.6 of the population of the United States was urban, 51.4 per cent. now live in cities. In other words, urban population has almost doubled in forty years, while rural population has almost been cut in half. A third principle which at least statistically argues for a further increment of mental disease is that the rate of mental disease is higher among inferior stocks than among superior. It is generally conceded that in late years the chief decline in birthrate has been greatest among superior stocks. Pollock is optimistic concerning favorable counteracting influences, among which he includes prohibition of liquor traffic, the movement to check syphilis and possibly more secure economic conditions. Furthermore, psychopathic hospitals, mental clinics, psychiatric social work and mental hygiene measures are apt to become more and more effective.

STRECKER, Philadelphia.

CHANGES IN NEUROGLIA CELLS RELATED TO AGE. ERNST BLUM, Schweiz. Arch. f. Neurol. & Psychiat., Festschr. f. Constantin von Monakow **13**:99-108, 1923.

One of the greatest difficulties in neuropathology is to determine whether an observation is to be considered pathologic or not. This is particularly true as it applies to the involutional changes of the cells and fibers noted in advanced age. It is true that some observers would regard changes occurring in the normal senium as pathologic. This, however, is incorrect as these changes are not accompanied by changes in function clinically.

In glia cells poor in cytoplasm, such as the satellite cells, one may note an increase in number. It is difficult to say whether this increase is dependent on senile changes or on morbid changes present at death. Alterations in the nuclei that accompany senility are deep staining qualities, elongated forms with excrescences, departure from a round to the polymorphonuclear shape. All told there is little that is characteristic of senility in this type of glia cell.

In the plasmatic glia cells, it was immediately noted that the basophil-metachromatic bodies described by Casamajor could not be demonstrated. A negative observation, however, is less convincing than a positive one. Blum found acidophil granules which he believed were related to fibrilogenesis. Nuclear changes were also found that were regarded as evidence of amitotic cell division. Sudan III preparations revealed lipoid granules, resembling the lipofuchsin granules of ganglion cells. The writer regarded these changes as pathologic and not normal for senility.

The fibrilogenetic cells of the marginal glial layer showed evidence of proliferation during senility. The nuclei were large and pale, the chromatin was granular. The cytoplasm was increased in amount; one could follow the arrangement of gliosomes into fibrils, the subsequent liberation of the fibril from the cell, and in the fuchsin-lichtgrun preparations, the disintegration of the cell plasm into small green granules. The fibers themselves were tough and knotty. Lipoid granules in great numbers were noted in the glia cells; there seemed to be a parallelism between the increase of lipoid in the glia and in the ganglion cells. This was interpreted as an independent chemical reaction going on in these two types of cells. In the outer layers of the cortex, occasionally one saw cells that appeared to be vacuolated as though the periphery of the glia cell plasm had been punched full of holes. Fat stains failed to stain these vacuoles. The nuclei were dark and suggested regressive changes. Increase in the number of corpora amylacea was noted, particularly in locations having glia cells rich in cytoplasm and active in fibrilogenesis. Presumably the cells gave rise to the corpora amylacea. The lipoid substances in the glia cells were apparently not incident to transportation processes. Blum believes that they are formed within the glia cells themselves and are characteristic of the normal senium. Glia fibers were not only increased in number but in thickness and in irregularity. Ameboid glia cells, apparently with regressive nuclear changes, appeared to exist normally. Senile plaques must be regarded as pathologic. It is true that they might be found normally under exceptional conditions, as in the case reported by Minea, in a 105 year old woman. Generally speaking, they are not a part of the normal senile process.

Often it is exceedingly difficult to determine whether a given process is to be considered as a normal product of involution or not. The general picture and particularly the clinical history must be taken into consideration as well.

WOLTMAN, Rochester, Minn.

INTRACRANIAL HEMORRHAGE IN THE NEW-BORN. WILLIAM SHARPE and A. S. MACLAIRE, Surg., Gynec. & Obst. **38**:200 (Feb.) 1924.

The authors present an interesting study of 100 consecutive cases of lumbar puncture in the new-born, ranging from four hours to fifty-three hours after birth. In this series, there were thirteen cases that showed definite signs of blood in the cerebrospinal fluid. It is interesting to note that the article takes up the various factors which may influence hemorrhage in the new-born, such as primiparae, multiparae, forceps, clotting time and syphilitic history.

In the forceps cases, eight in which low forceps were applied showed no blood in the spinal fluid; in the two cases in which medium forceps were required, both showed bloody spinal fluid. In the babies of syphilitic mothers, there were eight cases, two showing blood tinged spinal fluid. In six cases of prolonged labor, three showed the presence of blood by lumbar puncture.

In face presentations, there were three cases; one showing bloody spinal fluid.

The authors point out that the hemorrhage is probably due to rupture of the supra-cortical venous tributaries as they enter the sinuses, due either to the over-riding of the cranial bones or to tremendous pressure and extreme dilatation of these veins. Hemorrhagic diseases do not play as important a part as once was thought. In no case in this series was the coagulation time prolonged. The authors advocate treatment by repeated lumbar puncture and spinal drainage until the blood becomes clear. They show that the condition is far more frequent than is usually realized, and is easily missed and permitted to remain undiagnosed unless careful observation is made. Slight drowsiness, apathy, few muscular twitchings, failure to nurse properly, feeble and irregular respiration are the usual symptoms. Paralysis may also be manifest, but this is not at all constant. When the conditions are allowed to go unrecognized, they are usually noted too late for helpful intervention. The mother notes that the child fails to sit up or walk; teething is delayed, mental impairment is present, and paralysis becomes more apparent. Cases closely simulating Little's disease, such as spastic diplegia, paraplegia or hemiplegia, may be entirely due to intracranial hemorrhage at the time of birth, and not to an agenesis as is usually thought.

The cases in which bloody cerebrospinal fluid was found are being followed up by the authors to determine whether eventually mental or nervous symptoms will develop. In all these cases, lumbar puncture was sufficient to relieve the symptoms and obviated the more radical procedure or craniotomy which, however, is advocated by Holt. The authors warn against the use of lumbar puncture as a diagnostic or therapeutic measure in the presence of shock. The procedure has proved to be safe and effective in their hands.

TEMPLE FAY, Philadelphia.

FURTHER INVESTIGATION OF THE DEVIATION OF COMPLEMENT IN HERPES ZOSTER. ARNOLD NETTER and ACHILLE URBAIN, Compt. rend. Soc. de biol. **90**:461, 1924.

These investigations are of interest in that they indicate the identity of the causative agents of herpes zoster and chickenpox. This subject, from a clinical point of view, has been reviewed in America by McEwen (*Arch. Dermat. & Syph.* **2**:205 [Aug.] 1920), Kraus (*New York M. J.* **114**:162 [Aug. 3] 1921) and Riggs (*Minnesota Med.* **5**:646 [Nov.] 1922). All of these

authors, from analyses of clinical material, have felt that these two diseases resulted from either the same or closely related causes. It must be recalled that all cases of herpes zoster are not included, for example, those of syphilitic origin. The nonsymptomatic or essential variety alone is that in question.

Netter and Urbain have demonstrated an antigen obtainable from the serums and crusts of cases of herpes zoster (not labialis, which shows none of the reactions to be described) which fixes antibody of cases of herpes zoster. Further, they demonstrated that this antigen had the same action on blood serum from cases of chickenpox, and that antigen obtained from chickenpox crusts also fixed both chickenpox and herpes zoster antibody (*Compt. rend.*, January 26, 1924). They confirmed this in thirteen additional cases of zoster. They conclude from this, that the crusts or scabs of zoster contain an antigen capable of deviating complement of the blood serum of both chickenpox and herpes zoster; and that the converse obtains as well.

They cite examples of zoster being transmitted as chickenpox to others in three of their cases: (1) chickenpox in a daughter thirteen days after the appearance of zoster in the mother, (2) chickenpox in a student sixteen days after visiting a friend who had herpes zoster and (3) chickenpox in a man fourteen days after its appearance in his wife. The sequence—chickenpox herpes zoster—is much less common than the reverse, as shown not only by these cases, but also by a review of other reported cases.

The addition of this bacteriologic evidence to the clinical evidence already accumulated emphasizes the probable identity of the causes of chickenpox, and many cases of nonsymptomatic or essential herpes zoster.

KRAUS, Paris, France.

PATHOGENESIS AND TREATMENT OF SO-CALLED CONGENITAL CEREBRAL HERNIA. F. M. LAMPERT, Surg., Gynec. & Obst. **38**:159 (Feb.) 1924.

The author looks on this condition as an ectopy of the brain, of rare occurrence; the instance is but two or three cases in every 10,000 new-born children. Reports of two cases in which operation was performed accompany the article.

The first case was that of an 11 month old child with a large pedunculated outgrowth in the occipital region. The tumor proved to be cystic, and a small bony opening revealed its contents communicating with the cranial cavity. The bony defect was closed, with the eventual recovery of the child. The etiologic facts in this case are listed as:

1. Dystopy of the brain elements.

2. Incomplete closure of the skull because of the mechanical obstacle brought on by the dystopy.

3. Protrusion of the meninges with formation of a meningeal cyst partly because of disturbance of lymph and blood circulation and the consequent increase in intracranial pressure.

The diagnosis in this case was hydrochepalomeningocele occipitalis inferior.

The second case is that of a boy 2 years and 6 months of age, with a tumor at the base of the nose. The tumor was attached to the nasal bone; it was soft, at some points transparent, and at others pulsating. Compression did not reduce the tumor. It was, however, very painful. "The operation showed the tumor to be located at the central region of the orbit and separated from the bone. The nasal bones at their base were broken through and turned

backward. The tumor which extended into the skull cavity could not be removed and was resected at the base. The child died three days after the operation." The growth (microscopically, glioma) should be looked on as a dystopy—that is, as the result of incomplete separation of the cranial layer from the medullary and a coincident defect in the cranial bones.

The author shows the mortality, through others works, to be very high (50 to 60%). The prognosis is that of idiocy, paralysis, blindness, hydrocephalus, optic atrophy, succumbing in early childhood.

The author concludes that conservative treatment of encephalitis does not give favorable results; if operation is indicated, it should be performed within the first months of life. It consists in radical excision of the tumor and in the use of the osteoplastic flap as well as in the creation of conditions favorable for sufficient and continuous absorption of the excess of the cerebrospinal fluid for regulation of the intracranial pressure.

TEMPLE FAY, Philadelphia.

SOME REMARKS ON THE SUBCONSCIOUS. E. CLAPARÈDE, Schweiz. Arch. f. Neurol. u. Psychiat., Festschr. f. Constantin von Monakow **13**:200-205, 1923.

The term unconscious entered psychologic discussions about thirty years ago, since when it has been defined, its usefulness questioned and even its legitimacy contested. It has been used in three different ways: first, as an adjective, simply to signify that which is not present in consciousness, without judging whether the thing which is not present in consciousness is or is not in itself conscious. Thus the thoughts of my friend Pierre are unconscious for me the same as those of my dog. The functions of my liver and of my kidneys are unconscious, that is, I have no consciousness of them. Second, it has been used to signify that which possesses no consciousness. In this sense my watch is unconscious of its tick-tack, and certain authors claim that animals are unconscious. Third, it has been used substantially to designate a principle which takes account of the finality of vital phenomena, and among certain psychologists, like Freud, to designate the ensemble of ideas and affective memories, outside of consciousness, which nevertheless enjoy an independent rôle in the personality. This last position, Claparède thinks should be pitilessly repudiated, because it constitutes a contradictio in adjecto and says precisely the opposite of what it is wished to say.

The hypothesis of a subconscious is not contradictory and is perfectly legitimate. It is said sometimes that latent memories are subconscious when they are not actualities in an active memory. This is superfluous. In the same manner, the consciousness has been invoked to account for certain perceptions which at first rest unconscious but which nevertheless subsequently determine certain associations, as for example when we are suddenly struck with an idea without knowing why or look at an object without seeing it. Here also a physiologic explanation would suffice in most cases. In brief, the term subconsciousness is only admissible or useful when one applies it to a mental activity which takes place outside of the ordinary consciousness of a subject, an activity which implies a more or less distinct personality of an ordinarily conscious person. We can easily imagine an automaton who will respond to a tap on the patellar tendon or whose pupil will react to light, or who will write several words and dip its pen into the ink. But these words are always the same and the gesture is the same and does not vary according to the circumstances of the moment.

At the present time, without doubt, the idea of unconsciousness would seem to be superfluous. The reason of consciousness escapes us, but its manifestations do not.

WOLTMAN, Rochester, Minn.

THE HYPOGLOSSAL NERVE IN AN EMBRYO OF ERINACEUS EUROPAEUS. H. BERKELBACH VAN DER SPRENKEL, J. Comp. Neurol. **36**:219, 1924.

The hypoglossal nucleus of the European hedgehog was found to be the sole nucleus of origin of the genuine hypoglossal nerve fibers. The nucleus may be divided into an anterior one-fifth whose cells are visceral motor in type and which sends fibers to the geniohyoid muscle; a middle two-fifths which sends fibers from its dorsal portion to the hyoglossus muscle and from its ventral portion to the styloglossus and genioglossus muscles; and a posterior two-fifths which sends fibers from its dorsal portion to the longitudinalis linguae and from its ventral portion to the transversalis linguae muscle. The cells in the middle and posterior units resemble those of the anterior horn of the spinal cord with which the hypoglossal nucleus is continuous. There is no decussation of the emerging root fibers of the hypoglossal nerve. Although the fiber bundles from the various centers of the nucleus shift their relative positions within the nerve trunk along its course, they at no time interchange fibers.

The muscles of the neck which are innervated by the so-called "ansa hypoglossi," are localized in the caudal portion of the twelfth nucleus in the following order: rostrocaudally: thyrohyoid, sternohyoid, sternothyroid and omohyoid muscles. The motor innervation of the ansa muscles comes exclusively from the first and second cervical nerves; the ansa hypoglossi contains no true hypoglossal fibers. Sensory fibers, whose cells of origin probably lie in the spinal ganglion of the second cervical nerve, were seen to pass by way of the ramus descendens of the twelfth nerve to the tongue muscles, thyrohyoid and styloglossus muscles. They probably also supply the rest of the ansa muscles. The muscles in this region receive fibers from the superior and inferior cervical sympathetic ganglia.

A small group of cells, Roller's nucleus, lying medial and caudal to the nucleus of the twelfth nerve sends fibers along the twelfth nerve to ganglion cells located about the blood vessels. This nucleus is thought to be a vasodilator center.

GRAY, Chicago.

MANAGEMENT OF INJURIES TO THE CRANIUM AND ITS CONTENTS. JOHN O. BOWER, Ann. Surg. **78**:433 (Oct.) 1923.

The author discusses the treatment of cranial injuries with special reference to the management of cerebrospinal pressure. The use of lumbar puncture, with a spinal manometer, is advocated, and when the pressure reading is high, fluid should not be withdrawn except under carefully regulated readings, and then only small amounts to insure gradual reduction.

He advocates operation if, after eight hours, there is an increase to 20 mm. of pressure or more and no improvement has been noted. Blood pressure, pulse and respiratory findings are considered of exterme importance. The still, deep respiratory movements of compression denote pressure and should be differentiated from the superficial breathing of a shocked patient. Examination of the eyegrounds and careful history are two other important factors.

Cranial defects are taken up, the repair by means of an osteoplastic transplant being discussed. The article is well illustrated with cases from the Army

service. The method elaborated by Dr. Frazier is used with decidedly beneficial results and consists of freshening the bone edges of the cranial defect, obtaining a pattern of the bone and removing a thin layer of the outer table from an adjacent portion of normal skull, the pericranium to be carefully preserved on the transplant and sewed to the edges of the opening.

His series contained sixty-two cases; forty-six patients had acute cerebral lesions associated with fracture of the cranium which were demonstrated either by the roentgen ray, operation or postmortem. Fifty-two per cent. were operated on. Of the operative cases, there were eight fractures of the vault with no deaths: sixteen fractures of the base and vault with eight deaths; six operations for defects, no deaths. Of the twenty-two nonoperative cases, there were seven fractures of the vault, no deaths; thirteen fractures of the base, two deaths; fractures of the base and vault, two, with two deaths.

TEMPLE FAY, Philadelphia.

A STUDY OF THE INNERVATION OF THE TONGUE MUSCULATURE WITH PARTICULAR REFERENCE TO THE PROPRIOCEPTIVE MECHANISM. ORTHELLO R. LANGWORTHY, J. Comp. Neurol. **36**:273, 1924.

Muscle spindles were demonstrated in the extrinsic tongue musculature of pig embryos, and of adult cats, dogs, opossums and rats. Bilateral section of the twelfth nerve in cats produced complete paralysis of the tongue, with degeneration of the muscle spindles. Unilateral section of the twelfth nerve showed that there is some overlapping of the hypoglossal innervation in the midline. Section of the ninth or lingual nerve had no effect on the coordination of tongue movements in these cats or on the histologic picture of their muscle spindles. It is probable, therefore, that proprioceptive impulses pass from the tongue to the central nervous system by way of the homolateral twelfth nerve. In many specimens, small ganglia were found along the posterior roots of this nerve. The cells of these ganglia are sensory in type, and exhibit chromatolysis after section of the nerve. The number of cells in these ganglia is too small to account for all of the muscle spindles seen; again ganglia were not found in all specimens. Therefore, there are probably other sensory cells whose fibers run in the twelfth nerve.

GRAY, Chicago.

MENINGES OF BIRDS, WITH A CONSIDERATION OF THE SINUS RHOMBOIDALIS. O. C. HANSEN-PRUSS, J. Comp. Neurol. **36**:193, 1924.

The meninges of birds were found to be similar to those of mammals; namely, a dura mater, an arachnoidea and a pia mater. The spinal epidural space is not continuous because at places the dura seems to fuse with the periosteum of the vertebrae. Although birds possess a functional subarachnoidal space, it exists as a series of discontinuous spaces. Approximation of the pia and the arachnoidea at the borders of the sinus rhomboidalis, a space formed at the level of the sacral enlargement by a separation of the posterior funiculi of the spinal cord, separates the subarachnoid space from the meshes of the sinus. These meshes consist of a dense network, similar to that found in the subarachnoid space, the interstices of which contain a colloid-like substance. Anatomically these meshes appear to represent the subarachnoid tissue in the sinus. Since the central canal of the cord passes through the sinus unchanged, the sinus rhomboidalis cannot be regarded as a sacral ventricle.

GRAY, Chicago.

RECKLINGHAUSEN'S DISEASE—LARGE NEUROFIBROMA OF THE TONGUE. EGAS MONIZ, Rev. neurol. **30**:222 (March) 1923.

The paper is a short case report of polyneuroma in a man 48 years of age, a farmer, who presented the association of a plexiform neuroma and of a generalized neurofibroma. The two disorders often show points of similarity. They may be regarded as nothing other than modalities of neurofibroma.

In the case reported the patient presented a plexiform neuroma attached to or suspended from the right temples and right eyelid, and hanging as low as the shoulder. The neuroma was hard, pigmented and had an elephantine aspect. It was not painful on palpation. The patient began to lose sight in his right eye at the age of 9. When 14, he could see nothing with the affected eye. At 20, small tumor masses began to appear over all surfaces of the body. About this time his tongue began to develop a tumor mass, and this grew to the size of a hen's egg. Tumor of the tongue (or neurofibroma) is exceedingly rare. The case under discussion also showed another striking feature, namely, a definite loss of bony structure in the right temple. Neurofibromatous conditions with skeletal changes are more frequent in men than in women and are often hereditary. The present case is not hereditary. The disease certainly depends on a congenital predisposition, the mechanism of which is not yet known to us.

JONES, Detroit.

AFFERENT AND EFFERENT PATHWAYS IN AN ABDOMINAL SEGMENT OF AN INSECT. WILLIAM A. HILTON, J. Comp. Neurol. **36**: 299, 1924.

The structure of the abdominal ganglia of the larvae of the beetle, *Dendroides,* was studied in living and fixed preparations. Each ganglion possesses four branches: (1) a large dorsal branch containing afferent fibers from bipolar cells lying at the base of the dorsal sensory hairs; (2) an afferent branch from the bipolars of the ventral hairs, but containing in addition one division from bipolar cells lying within the muscles (probably muscle sense); (3) a motor branch to the muscles of that segment; and (4) a dorsal branch which runs cephalad to supply the spiracular openings. A number of the processes from the dorsal and lateral hair bipolars run to the ganglion of that segment but turn immediately to pass cephalad. In any branch to a ganglion there is some evidence of fusion of the processes of the motor cells because a single muscle cell appears to be innervated by about thirty neurons. No evidence of similar fusion was found in the case of the sensory cells. In addition, each abdominal segment contains nerves which come from more cephalic parts of the central nervous system and which supply the intestines and kidneys. These may possibly be analogous to the sympathetic fibers in higher forms.

GRAY, Chicago.

INTRACRANIAL PAIN. JOHN F. BARNHILL, Surg., Gynec. & Obst. **38**:210 (Feb.) 1924.

The author points out that the dura is the only intracranial tissue that is to any degree endowed with sensation. All intracranial pain, he feels, is due either to pressure on or injury to the dura, or to toxic products which irritate the nerve terminals either directly, as in abscess, or indirectly by way of the blood stream.

Intracranial pain is usually due to infection of the paranasal sinuses and the mastoid cellular labyrinths. This may cause increased intracranial pressure

along with other toxic manifestations and give pain by pressure on the dura. He considers that tic-doloreaux may cause intracranial pain, although usually its symptoms are referred to the periphery and are probably reflex. "Clinical experience and dealing surgically with the disease, leads to the conclusion that both the disease and its terrifying pain are in reality inside the skull."

Tumors of all kinds causing intracranial pain do so by their pressure on the dura directly or through increase in intracranial pressure. He concludes that intracranial pain is undoubtedly less frequent today than formerly.

TEMPLE FAY, Philadelphia.

THE NUCLEUS ISTHMI OF THE FROG. O. LARSELL, J. Comp. Neurol. **36**:309, 1924.

The nucleus isthmi of the frog's brain consists of a cortex of closely packed cells and a medulla containing neurons and fibers. At the ventrolateral border of the nucleus, fibers enter through the hilum to terminate about the dendrites of the cortical and medullary cells. The nucleus isthmi receives afferent fibers from the lateral lemniscus, the optic tectum and the inferior colliculus. It sends crossed and uncrossed fibers to the optic tectum, commissural fibers to the nucleus of the opposite side both by way of the optic chiasm and the anterior medullary velum, and efferent fibers from the cortical cells which could be traced as far as the thalamus. From its connections, the nucleus isthmi appears to be homologous with the medial geniculate body of mammals.

GRAY, Chicago.

STUDIES ON THE RETINA. THE IDENTITY OF THE DEVELOPING VISUAL CELLS IN AMBLYSTOMA LARVAE AS REVEALED BY THEIR RESPONSES TO LIGHT. S. R. DETWILER, J. Comp. Neurol. **36**:113, 1924.

In the early stages of its development the retina of Amblystoma contains two types of conical elements: (1) large cells whose nuclei project through the external limiting membrane, and (2) small cells whose nuclei lie below this same membrane. In light adapted retinae of larvae whose eyes had just begun to function, an elongation of 7.3 microns was observed in the myoid of the small conical cells, without any corresponding change in the myoid of the large conical cells. In eyes adapted to the dark, a migration of the epithelial pigment of from 13 to 14 microns was observed. Their behavior indicates that the large conical cells are the progenitors of the rods and that the small conical cells are the progenitors of the cones of the definitive retina.

GRAY, Chicago.

MONGOLIAN IDIOCY IN BOTH TWINS. AUGUST STRAUCH, J. A. M. A. **81**:2181 (Dec. 29) 1923.

The parents of the twins affected with mongolian idiocy were healthy, intelligent and without any inherited taint. The mother at the time of conception was 20 years of age, and the father, 30 years and six months. The occurrence of a mongolian idiot with a normal twin has been observed in double ovum twins, as far as known. Mongolism in both twins has been described only in twins of the same sex. They are probably single ovum twins. Strauch believes that the conception of mongolism being due to an endogenic factor seems to find support in these observations.

NIXON, San Francisco.

Society Transactions

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

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C. M. CAMPBELL, M.D., *President, in the Chair*

INHERITANCE OF MENTAL DISEASES. DR. ABRAHAM MYERSON.

The term insanity as well as the term feeblemindedness and all terms which imply that there is necessarily a unitary character to psychiatry are more than useless—they are pernicious in that they exercise an influence on the mind, which vitiates most of the work that has been done on the inheritance of mental diseases. There are independent groups of mental diseases which will be declared to have no hereditary value and others to run in families, as the phrase goes. That this "run in families" is necessarily hereditary, to be compared with mendelian heredity or the transmission of the major biologic qualities, seems to me not to be the case. The working hypothesis that wherever mental diseases appear to run in family groups some factor is at work which has been introduced from the environment and which has injured the germ plasm, is here advanced. This theory has been called blastophoria by August Forel, so that we may speak of this working hypothesis as the blastophoric concept.

There is no unity in feeblemindedness; the problem of the cretin belongs to the problem of endocrinology; the problem of the congenital syphilitic is the problem of syphilis; the problem of spastic diplegia and other spastic states, so often associated with idiocy, is a problem of trauma at birth, infectious disease, and the like. Occasionally it represents a failure in development, and there it is part of the problem of teratology and has no especial relationship to other types of feeblemindedness.

The conditions mentioned in the foregoing rarely have anything to do with heredity, or at least there is no obvious connection with defect or disease in an ancestor. They tend to be sporadic, to occur in families which cannot be differentiated from the ordinary groups in the community.

There also occur, especially among the more markedly defective, the idiots, cases in which nothing can be found that adequately explains the occurrence of the defect. As one studies the cases of marked defect, one is struck with the large number in which this represents a variation from the family level and without any hereditary background. I have as yet had no definite research experience with the higher grades of feeblemindedness, the moron type, which constitutes probably the largest group of the feebleminded. The data on which these are declared to be hereditary in origin, which appear in the textbooks and in the special writings of Goddard and Davenport, seem to me to be extremely faulty. Especially is there to be criticized as unscientific such family studies as "the Kallikaks" in which a field worker on the basis of hypothesis, guess, and intuition declares the dead and the quick feebleminded without the slightest definite reason for so doing. Making a diagnosis of moronity is a clinical matter attended with difficulties, but the field workers of Drs. Goddard

and Davenport have no difficulty in doing this on the basis of a court record, of a tradition, and of a cursory glance. That there are groups of the feebleminded in which the disease runs in families over one or more generations seems to be without doubt. How large a proportion this constitutes cannot be stated at present. It is unquestionably much smaller than has hitherto been assumed, and even in the cases in which it exists is not necessarily a fixed character persisting from generation to generation. It is interesting to note that the families founded by definitely known feebleminded persons discharged from Dr. W. E. Fernald's institution have not shown the inevitability of feeblemindedness in their descendants nor any trace of the much discussed prolificity which appears so conspicuously as a bogyman in the semiscientific and lay literature.

The Situation in the Psychoses.—There is no evidence to show that general paralysis depends on any hereditary factor. There is no evidence that the arteriosclerotic psychoses depend on anything but the appearance of arteriosclerosis in the brain, which seems to have the same background as arteriosclerosis in general. Cerebral arteriosclerosis occasionally occurs in members of the same family and an arteriosclerotic ancestor may have descendants with mental disease. These are mere coincidences, for it has been shown by Koller, Diem, Jolly and others that cerebral arteriosclerosis occurs more frequently in the ancestors and collaterals of the "sane" than the "insane." There is no scientific evidence that alcoholic mental disease is hereditary. Many of those who become alcoholic are peculiar, but the main basis of the alcoholic mental disease is alcohol operating in a curious manner on an individual. This idiosyncrasy to alcohol is a pharmacologic matter, to be compared to the reaction to bromids or morphin of certain persons. It has no distinct relation to psychopathy except that psychopathic persons often drink to excess. The study of Elderton, Pearson, and others is interesting in relation to this matter.

The foregoing three groups account for about 40 to 50 per cent. of all patients admitted to hospitals for the insane, and there is therefore at least that percentage in which no valid evidence has been brought forth to show the working of any force which can be compared to heredity. I come to a group of mental diseases which by all accounts and from all the literature appears in family groups—sometimes in several generations and sometimes in several members of the same generation.

Before taking up the situation in dementia praecox, manic-depressive psychosis and the involution diseases, a few statements in regard to certain data collected from the literature and especially from the work done by Mott in London and by myself in the Taunton State Hospital will be of interest. First, over 10 per cent. of the inmates of the state hospital at Taunton are related to one another closely. The largest groups of those related are the brother-sister group. The other groups in order are: sister-sister, brother-brother, mother-daughter, mother-son, father-daughter, father-son, aunt-descendant, uncle-descendant, cousin groups. Throughout the literature it appears that there are more females in the family groups with mental disease than males, and that so far as can be discovered by figures, the female transmits mental disease more frequently than the male. This, however, can be explained by certain facts conspicuous in the Taunton cases. The female insane have a much higher marriage rate than the male insane, especially in the dementia praecox group; the marriage rate of those with general paralysis and senile dementia is about equal to the normal marriage rate; the marriage rate of the male dementia praecox patient is extremely low, whereas the marriage

rate of the female dementia praecox patients is suprisingly higher than that of the male, yet distinctly less than that of the average female. Because the female in the group in which mental disease tends to occur marries more nearly in accordance with the normal marriage rate than does the male, she is more liable to have descendants and thus more liable to transmit mental disease. Again, the male of a family is much more apt to wander away from the district in which he was born than the female and thus to appear as a sporadic case in the statistics of some other institution. These facts are social rather than biologic in nature, and there is no conclusive evidence that the female is more liable to transmit mental disease than the male.

It also appears from the data gathered all over the world that the descendant (in family mental disease) is apt to have the mental disease at an earlier stage than does his ancestor and in a worse form. This phenomenon is called anticipation and has been greatly stressed by Mott and others. While it is true that there does appear to be some basis for the opinion that there is this tendency, it also appears that the way the statistics have been gathered explains part of the situation.

A survey of the world literature reveals only one case in which mental disease is actually known to have persisted for four generations, and that is in the case reported by myself. There are few cases compared to the total bulk of mental disease in which the mental disease occurs for three generations. Most of these also have been recorded among the Taunton cases. It appears probable that mental disease does not occur frequently for more than two generations. Oddities and peculiarities may persist, but difficulty in evaluating these or in tracing them makes it impossible to give them any great scientific value.

(*a*) If dementia praecox occurs in an ancestor and if his descendant has a mental disease, that mental disease is most likely to be dementia praecox. This opinion is based on a survey of the literature and of my personal studies. Occasionally, other mental diseases will occur in the descendant, but in so scattering an incidence as to be unimportant. It is quite noteworthy that there is a distinct tendency for the disease to manifest itself in a more serious form in the descendant and in greater liability to early dementia. (*b*) If manic-depressive psychosis occurs in an ancestor, the "insane" descendants seem to group themselves in two classes: the larger group which tends to be manic-depressive, and the smaller which tends to be dementia praecox. Most authors concur in this statement, although some state that dementia praecox is rare in such descendants. It may be stated that typical cases of manic-depressive insanity in an ancestor are followed by manic-depressive insanity in the descendant. The atypical cases, those in which there is some doubt as to the diagnosis, are followed by dementia praecox in the descendant. (*c*) Involution psychosis in an ancestor, if followed by mental disease in a descendant, is usually succeeded by dementia praecox. There is a surprising unanimity of experience and opinion on this point. (*d*) If there is paranoic mental disease in an ancestor and the descendant has mental disease, he is liable to have either paranoic mental disease or dementia praecox. It will thus be noted that in these cases there is some tendency for the psychoses to be dissimilar in clinical type and for the psychoses to trend toward dementia praecox in the descendant.

The Psychoses of Brothers and Sisters.—The psychoses of brothers and sisters tend to be alike. Dementia praecox in a group of siblings is apt to be uniform in its manifestations, although one classical case has been reported and personally observed in which there was a remarkable transition in type

from what appeared to be pure paranoia to hebephrenic dementia praecox in five sisters. Where manic-depressive insanity occurs in a sibling group, the manifestations are closely alike. It sometimes appears that one sibling becomes insane at the involution period while another becomes insane early in life. Nevertheless, although one will be called involution disease and the other perhaps dementia praecox, a close study will reveal the fact that the two psychoses are fundamentally alike. There are a few cases recorded in which manic-depressive psychosis and dementia praecox occur in the same sibling group. On studying them I have not been able to convince myself that typical cases of manic-depressive insanity and dementia praecox occur in the same sibling group. It is, however, stated by some authors that this is possible and does occur. If so, it is, relatively speaking, rare.

Normal and Abnormal Heredity.—Extensive studies made by Koller, Diem and Jolly indicate that certain characters are of no fundamental importance in determining psychopathic heredity. For example, it appears from the work of these three authors that neurasthenia and psychoneurosis, generally speaking, occur as frequently in the ancestry of the so-called normal person as in the ancestry of the abnormal person. It also appears that organic brain diseases are more common in the ancestry of the normal than in the ancestry of the abnormal person. This is especially true of cerebral arteriosclerosis, hemiplegia, apoplexy and the like, which are often given great weight in the literature. Certain types of alcoholism seem to occur somewhat more frequently in the ancestry of the abnormal than in the ancestry of the normal person, but not to a striking degree. Mental disease in an uncle or aunt appears as frequently in the history of the normal as in the history of the abnormal person. Mental disease in a father, mother, grandmother or grandfather occurs very much more frequently in the ancestry of the abnormal than in the ancestry of the normal person. Such diseases as cancer, tuberculosis, diabetes, and the like, seem to me to have no psychopathic bearing. Tuberculosis occurs frequently in the history of the ancestors of the feebleminded in institutions, but this is because tuberculosis is frequent in the social stratum from which these people come. At Dr. Fernald's institution in Waverly there is less tuberculosis than in the general community, largely because of the good hygiene of the institution. Therefore, much of what has passed as psychopathic heredity, nervousness, tuberculosis, diabetes, organic nervous disorders, and to a limited extent, alcoholism, has no psychopathic bearing. This is also true of much mental disease. The fact that an ancestor had had senile dementia or an arteriosclerotic psychosis has no bearing, so far as can be discovered, on the dementia praecox, feeblemindedness, or other psychotic manifestations in a descendant.

A Consideration of Certain Theories of Heredity.—Polymorphism, sponsored by Esquirol and Morel, and developed by Lombroso and a host of other writers, postulates that all forms of mental aberration from eccentricity of character to cretinism, including all of the psychoses, neuroses, epilepsy, and every departure from a hypothetical normal, are manifestations of the psychopathic constitution; that the disease condition manifests itself in a slight form in one generation and then increases degree by degree until, when a fourth generation is reached, the stage of idiocy appears and the stock then disappears. This remarkable theory which still shows its effects in all writings on the subject must be evaluated according to the time in which it originated. Not only may we say that information was lacking at the time when this formula for heredity was promulgated, but we may say also that the formula is never observed in operation. The only four-generation group that has been dis-

covered is the group I have described in which no such transition occurs. On the whole, it may be stated that polymorphism in the main may be rejected, although there is some evidence of a limited type of transformation and a limited linking together of various mental states.

Of late years biologists have introduced the concept of mendelian heredity into psychiatry. Mendelianism in psychiatry can by no possibility be proved at the present stage of psychiatric knowledge. It is based on polymorphism.

The Blastophoric Theory of Forel.—Forel states in opposition to what is usually considered true weismannism that the environment in the form of toxic substances, and especially alcohol, may so alter the germ plasm as to produce defective individuals, and that this defect may persist for generations. As a matter of fact, it may be stated that it has not been shown that alcohol, or syphilis for that matter, alters family groups in a permanent way or more than one generation of individuals. The effects of alcohol in an ancestor have not been studied in the case of man; but there is no reason at present to doubt that the environment may alter generations of individuals in a more or less permanent way. To corroborate this point of view the work of Stockard and his colleagues and of others may be cited to show that, irrespective of the problem whether or not acquired characteristics can be inherited, the environment may be so brought to bear on the individual as to alter his germ plasm, and this in a manner that persists for several generations. It is important to know that experimental science has substantiated the opinion that what happens to an ancestor may, and in certain instances does, alter the qualities of the descendant. This is not really opposed to weismannism.

If it comes to a choice between the present attitude taken by psychiatry toward mental diseases and an attitude which denied that there was any inheritance of mental diseases, I should favor as more true and more conducive to progress, the latter. The first view applies to a limited number of the types of the psychoses, and even in the case of these does not explain the origin of familial mental disease. The present view leads to pessimism and the adoption of a policy that does not aim either at investigation of causes or at therapeutics. Psychiatry needs to investigate the inheritance of mental diseases and needs to separate out the groups, to study each group from every angle, and to keep in mind that seventy-five years ago the leading medical men of the world were as firmly convinced of the inheritance of tuberculosis as psychiatrists today are of the inheritance of most mental disease.

DISCUSSION

PROF. G. H. PARKER: It is hazardous for a biologist to speak to a body of experts on a subject they know so well and that he knows so poorly, but we can all agree on methods of investigation. I know of nothing more depressing than to see a young person who has had three or four weeks' experience in some laboratory turned out on the community to learn the nervous composition of the people of the community. This is surely an unscientific method. There is only one kind of science in medicine, as in biology, and that is good science. We must keep away from all other kinds.

The whole question that has been raised by Dr. Myerson about inheritance gives you some idea of what the biologist is facing. The question is no more complicated in man than in mice or in guinea-pigs. The mendelians have attempted to divide their material on the basis of unit characters. This is quite artificial. Different eye colors may be taken to represent such characters, but eye characters are so abundant that they merge one into another and cannot be

distinguished, probably because they are really not separable. It is true that an enormous flood of light has come with the mendelian movement, much beyond what could have been expected twenty-five years ago, but it is well not to be carried away too easily.

It is true that the human being is enormously complicated, and that he presents a difficult problem, but I would not have you think from what Dr. Myerson has said that we know earthworms any better than we know human beings. The ameba is transparent, but in another way it is not transparent. It is as much a puzzle and a perplexity as the human being is. Inheritance is an operation that is puzzling and complex because of its many sides; human inheritance illustrates this very well. There is the social side, and human society is enormously complex. Man is peculiar in that he teaches. Very few animals teach. In this respect, man is unlike almost all other organisms. This involves a kind of inheritance that we do not encounter in lower forms. The human being, like every other organism, is always doing something. Where do these capacities come from? How much is inherited, and how much drawn from the environment? I am talking to you. How much of this process is inherited? I have a vocal organ with muscular and nervous parts that have come to me through my germ plasm. In my speech lies simple inheritance, and I do not doubt that many of the elements are mendelian. The words I am using are not inherited. I picked them up in my social relations. In the simple operation of talking to you I am exhibiting an immense amount of organic inheritance and a corresponding amount of social inheritance. Both normal and abnormal persons are exhibiting this double inheritance continually. There is a certain amount that is organic and a certain amount that is social, and these two are so intermingled that it is simply impossible to separate them. In a broad way we are bound to admit a large amount of organic inheritance in every act, while social inheritance also plays an important part.

Dr. Myerson has touched on the vexed problem of the inheritance of acquired characters. The acquired character is a trait that results from some direct change in the body not received through the germ. It is puzzling to make a distinction between organic inheritance and social inheritance, from the standpoint of acquired characters. The inheritance that comes through the egg or sperm is strictly organic, but it is doubtful that acquired characters are passed on in this way. Examples are difficult to find; in fact, we do not know that the inheritance of acquired characters in man really takes place. A protozoan is a single cell. The single cell is both body and reproductive element, because in reproduction the body divides and its substance goes directly on to the next generation. Inheritance in a protozoan may include the inheritance of an acquired character. This also happens in nonsexual reproduction. A sea anemone simply divides in two as a protozoan does, but in the higher animals, snails and clams, crabs, insects, and in animals with back-bones, such a method does not occur. They do not reproduce nonsexually. The influence of the environment on the germ in higher animals was much discussed in Weismann's day. Later it was admitted that external influences do affect the germ. Experimentation shows this conclusively. There is no doubt that there is influence of the environment on the germ, and one may have a feature thus produced handed down from generation to generation. Temperature changes and other such changes that penetrate the body may produce such alterations. Chemical substances in this respect are most important. The internal secretions affect the

germ. The study of alcohol has been extremely interesting, and Stockard's work has shown that germ cells may be profoundly influenced by the circulation of alcohol in the body. The parent is not so much influenced by the alcohol as the offspring are; infertility results and the stock dies out. Pearl has pointed to the interesting fact that weak germs are killed; in this respect, alcohol may be a selective instrument of value. But work on chickens is not work on man, and what applies to one may not apply to the other.

The last number of the *Journal of Experimental Zoology* has an article by Guyer on eye defects. Guyer has shown quite clearly, I think, that defective eyes can be produced in rabbits by the injection of lens materials in various ways into the pregnant female. These defective eyes are inherited in both the male and female lines. Those in the female lines may be said to be due to poisons carried through the placenta. But when this defect is inherited through the male the sperm cell must be the means of transmission to the next generation. This raises the question, What is an acquired character? How early in the development of the animal may it arise? The lens toxins which are handed on may be part of the living substance, and thus they complicate the question of what an acquired character is. I am not sure but that in Guyer's work we have what biologists have long looked for, but we may be learning a great deal about inheritance by these experiments without getting very far ahead with the old evolutionary problem from which they have sprung.

The question of the inheritance of acquired characters has been studied by many able men, and they are working toward something that may in time give us some positive evidence. The modification of the germ plasm by the environment also occurs. It is for the psychiatrist to work out many of these relations to human life. You have concentrated on one species of animal; we have spread over half a million.

Dr. William Healy: I think Dr. Myerson was hardly fair to the situation in regard to the Kallikak family. Those of us who have watched the material grow know that Goddard and Davenport did take into account a great deal of the criticism that Dr. Myerson offers. They felt, however, that if they could go into the community and so readily get reports of this nature concerning individuals, there must be much truth in the story of extensive mental defect, etc.; and even if discounted 50 per cent. there is a great deal of import in the reports. I am immensely critical of that type of work when general community report is the basis of diagnosis, but still I feel there is considerable to be said for the value of the efforts of these workers.

I remember years ago seeing a field worker at a meeting trace out criminalism and delinquency on a chart as being due to heredity, leaving out entirely the sort of environment that the delinquents were living in. I mean that bad housing, the influence of neighborhood spirit and bad companions, etc., were left out as possible causations affecting more than one generation. I regret very much to say that as a result of a field worker having been with me at one time material was published in a certain chapter of a certain book that I regard as quite unwarranted in its interpretations of heredity. I deplore any method by which we so readily pass judgment on the causations of human tendencies and psychopathic behavior because it leads to thoroughly unsound conclusions.

Dr. W. E. Fernald: Dr. Myerson and Professor Parker have shown some of the great difficulties in applying the theories of Mendel to the study of human

intelligence. It is a far cry from a study of white and red peas to the study of human intelligence. The study of human intelligence implies a complex combination of factors.

I have had some experience with social workers who have assembled material for eugenic research. These investigators, although liberally educated, had slight actual acquaintance with mental disease and defect. This lack of experience did not discourage them from diagnosing cases of supposed inferiority or mental disease occurring one, two, three, or even four generations previously; in fact, a man who lived over the mountain one hundred years ago was diagnosed as rapidly and certainly as one who is now alive. I proposed to one eugenic research worker that she begin with a certain admission number and study cases in order as they were admitted; she was not at all impressed by these "run of the mine cases" and I found in a short time that she was only interested in the cases where there were one, two, or more feebleminded brothers or sisters in the family.

My office is near the front door of this institution and puts me in close touch with the siblings, parents and collaterals of my patients. As I see them, these visitors represent a good cross section of our Massachusetts population. We are all familiar with the types of hereditary febelemindedness which are found in several sections of Massachusetts. No one doubts the existence of cases in which the present generation is feebleminded, and the father and mother as well. In my experience, however, these are the museum cases. In my institution it is difficult to collect a group of this sort; in fact, we have just lost the classical family of mother and eight children who served for so many years for teaching purposes.

The material for Goddard's book on feeblemindedness was found in central New Jersey, where the people described have been on an inferior moral, social, and economic plane for many years, and where they have intermarried for generations. I doubt if any such large group of similar cases could be found in Massachusetts.

Dr. Samuel Howe, the pioneer with the feebleminded in this country, made a survey of all the idiots in Massachusetts. He obtained this information from the town clerks in every town in the state. This survey was made in 1848. A few years ago we got in touch with the town authorities in many of these towns and were not able to find a single feebleminded person of similar name in any of these towns.

The study of mental heredity of human beings is difficult for many reasons, one being the fact that no observer can personally observe more than three generations. A few years ago I frequently lectured before women's clubs. At one of the meetings the lady in the chair said, "Please do not say anything about heredity. Every lecturer this winter has talked about heredity and our members have studied their ancestry with much dismay." So in the course of my talk I said, "I challenge any person here to go back three generations, including siblings, collaterals, and grandparents, without finding some instance of mental disease or defect." The members of the audience looked rather wildly at one another for a few minutes, but no one responded to my challenge. I believe this is a safe challenge in any similar group. The incidence of mental disease in average American stock is not at all understood. We very much need a study of the heredity of the average normal child as found in the public schools. The work of Goddard and Davenport was done with institu-

tional defectives, and they are largely of the hereditary type. The "accidental" defective without hereditary history is usually cared for at home, so that both Goddard's and Davenport's studies, even if one admits all they claim, were made on a highly selected group of persons. The class of parents who bring their children to the outpatient clinics and want advice represents a different social and mental level from that of the average committed defective.

In the public school mental clinics in Massachusetts, we are now examining about 5,000 children a year, most of whom are technically feebleminded. This group, of course, is entirely unselected except on the basis of their intellectual level. They seem fairly to represent the average American home; they come from the home of the well-to-do, of the middle class, and of the poor. Many of these children have no siblings or collaterals with intellectual defect.

Dr. O. J. Raeder: When I was studying medicine the professor of pathology began his opening lecture with the statement: "Pathology was the foundation of all medicine." Dr. Myerson with his iconoclastic housecleaning has attempted to rid some types of mental disease of the grime and dust of symptomatic grouping preparatory to their reclassification on a pathologic basis. He has mentioned how certain mental diseases have been so studied and so learned about through pathology. He has mentioned general paralysis and shown how it was taken out of the vague group of insanities and brought back into the domain of general pathology. So with some feeblemindedness, such as cretinism, and syphilitic hypophrenia, which some authors claim does not exist. In this way some "disgraceful" mental diseases have been brought back into the "respectable" fold of general medicine. This is important from the point of view of mental hygiene and therapeutic psychiatry. The mother, for instance, carefully guards her feebleminded or delinquent child from the common knowledge, due to a false feeling of disgrace about mental or moral disorders, instead of trying to find out why it is so. If such conditions are returned to the field of general pathology, the mother will readily bring her child to the children's hospital and so make it easier for the mental hygienist to examine and treat it.

Dr. E. S. Abbot: Dr. Myerson is to be congratulated on the sanity of his paper. We of this generation cannot solve the problem, as Dr. Fernald has said, so it is up to us to make such records as future generations can use in solving it. We should make such definite records.

Dr. C. M. Campbell: Dr. Myerson has made a most interesting presentation of a complicated problem. In his discussion he has referred to dementia praecox and to manic-depressive insanity as if they were definite disease entities. As a matter of fact, the present line of progress in psychiatric investigation is away from such a conception; one does not look on dementia praecox as an entity, any more than one looks on epilepsy or feeblemindedness as such. The analysis of a schizophrenic condition reveals a most complicated series of forces at work. There is much that points to the importance of the constitutional factor in the schizophrenic conditions, but there are many problems connected with the topic of deterioration. The genetic study of the personality is a fascinating study, and the recent suggestions of Bleuler indicate definite lines of investigation. Students of the heredity of mental disorders will have to consider the presence not only of individuals with well marked schizophrenic conditions or with pronounced schizoid personality, but also the possible existence of special groups of functions, the syntonic or cycloid and schizoid mechanisms, which may coexist in the same individual with varying preponderance of one or the other.

SOME NOTES ON PRIMITIVE IDEAS. DR. ELTON MAYO.

DR. MYERSON: Professor Parker, whom I expected would disagree with me, apparently agrees. It is a great comfort to know that some biologists are willing to believe that the environment can alter the organism so as to produce defect or disease which runs for more than one generation. If a character does appear in one generation, and has appeared for the first time in the previous generation, it is not necessarily a question of any specific form of heredity. The question may be simply this: What has injured the germ plasm to produce defect? Every group of people is concerned with the environment, not only as it alters or affects the individual himself, but as it alters the qualities of the next generation. It is not entirely improbable that chronic emotional states arising out of certain phases of the environment, or out of the reactions of individuals to environment, may alter germ plasm. Emotion is organic; it has a tremendous endocrinal and vascular component, and that endocrinal and vascular component may, according to our present knowledge, change germ plasm. This means that social inheritance, which Professor Parker so sharply separated from germ plasm heredity, is in some respects linked up with it; that social inheritance may possibly be a component of biologic inheritance. I am inclined to believe that there is no sharp line of demarcation between that external environment, which we classify as environment, and that internal environment which we call the body.

Dr. Healy began by rebuking me for my criticism in regard to the story of the Kallikak family, and then said we might discount 50 per cent. of the matter as due to exaggeration or misinformation. But 50 per cent. wipes out the difference between normal and abnormal heredity, as has been well shown by Kohler, Diem, and Jolly, as well as by every-day observation. Heron pointed out that Davenport's instructions to his workers were such that they could not fail to bring in what they did. This may or may not be so. At any rate, I think we can dismiss from our minds any number of Kallikak families. If true, they are museum cases, and therefore are not true examples of the bulk of feeblemindedness.

Dr. Fernald, because of his wide experience, has the most solid contribution to give, but he has neglected to speak of the only valid work on feebleminded-ness, and that is what he has recorded himself, as to the fate of the many people who have left his institution against advice, settled in the community, and in many cases, married. Of those who married, the greater number led normal lives and got into no difficulties. Some had feebleminded children; the most did not. Furthermore, and this is important in view of the "appalling fertility of the feebleminded," the families of these feebleminded did not average one person to a fimily. What is really meant is the fertility of low cultural level, not the fertility of feeblemindedness. Dr. Fernald has pointed out a fact of importance: Institution cases are different from those studied in practice. The practicing psychiatrist sees a large mass of cases which spring from normal stock and never reach the institutions.

Dr. Raeder has emphasized what I have tried to say, viz., that as we become better psychiatrists, as we get to know something about etiology and pathology, we shall get somewhere in our study of the inheritance of mental diseases. In this way, we may get to know the origin of dementia praecox, etc.

Dr. Campbell's description of what Bleuler is trying to do brings up the whole question of freudian psychiatry. I see nothing gained by changing the name of dementia praecox to schizophrenia, and I see nothing gained by classifying mankind as "schizoid" or "syntonic." Freudism is merely descriptive and analytic from a metaphysical point of view. At any rate, I see nothing in the

present freudian work which helps us in fundamental understanding of the genesis of familial mental disease. That may be my fault, but perhaps some other explanation can be found.

What we, as physicians, need to keep before our minds is that every human being is an organic mass and has an organic background. Mood and emotion are organic. Heredity is organic, and the environment is a mass of organic forces which penetrates within the individual, interplaying with the organic structures which we call in part somatoplasm and in part germ plasm. It is inconceivable to me that the organic forces of the environment have no affect on the organic forces resident within germ plasm.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, April 25, 1924

CHARLES BURR, M.D., *President, in the Chair*

HIGH POINTS IN PREVENTIVE PSYCHIATRY. DR. E. A. STRECKER.

Preventive psychiatry should be distinguished from mental hygiene. The latter concerns itself with many things that, strictly speaking, have little to do with prevention; the former should expend its efforts on reducing the incidence of mental disease. On what might be termed the somatic side, there are a number of outstanding opportunities. Syphilis, which is responsible for 15 per cent of all mental disease, is preventable and controllable. In some measure, the same is true of alcohol, habit forming, industrial and other exogenous toxins. Among the acute and chronic infections, influenza, epidemic encephalitis and tuberculosis merit particular consideration from the angle of prevention. Since the span of human life is gradually being lengthened, arteriosclerosis occupies a special position and constitutes a challenge to preventive psychiatry. Too many great brains break by reason of arteriosclerosis and too many constructive careers are brought to a conclusion by this disease.

Probably the golden opportunity for preventive psychiatry is to be found in influencing personality during its formative stage, that is during childhood. If mental catastrophe is to be averted in many instances, there must be sufficient mental space and opportunity, so that the personality of the child may grow by imitation, health-giving suggestion, satisfaction of curiosity and assistance in bridging the gap between self and socialization; in these is comprised the relinquishing of love of power, outlets for childish savagery, romancing, etc. Even after the personality has been determined, preventive psychiatry may still find opportunities. Southard indicated some of these when he described the five great evils—ignorance, poverty, crime, disease and legal entanglement—as producers of mental disease. A practical program of preventive psychiatry should review the crises of life, from adolesence to old age, and aim to modify and safeguard the indivdual against their dangers.

Heredity is relegated to last place on the program, not because it is unimportant, but because it cannot be controlled to any extent, and must remain for a long time at least a "fixed quantity in the equation" of mental disease.

SOME NOTES ON PRIMITIVE IDEAS. DR. ELTON MAYO.

In the history of philosophic thinking since Plato, the word "idea" has run through many meanings. It has been used to specify the universal logical form, the external stimulus to thought or the thought itself—the last being the usually accepted significance in our time. And all these meanings have from time to time been repudiated. Behaviorism, for instance, endeavors to formulate a psychology based on "action-patterns" or "conditioned" responses to the stimulus of environment. Yet, in spite of behaviorism, there is still justification for using the word "idea" in empirical descriptions of psychologic investigations. We may agree that the function of "consciousness" is to secure a closer adaptation of the individual organism to its immediate environment. We may agree also that behavior has always an "objective functional reference" (Holt). But we are forced at once to face the fact that an identical environment does not call forth the same behavior response in different persons. A wide range of variation of response is possible between unemotional recognition and hysterical terror. And in each case the response is adequate. An empirical psychology has to take account of the meaning for an individual of a given situation.

The word "idea" may be used tentatively to mark out this area of investigation. Psychology does not seek to warn the neurologist off this territory. But the hope of an adequate physiology of "integrated reflexes" cannot be allowed to hinder the direct investigation of individual situations. Neurology has everything to gain and nothing to lose by empirical inquiry into the relaton of "ideas" to conduct. And studies outside the physiology, psychology area—anthropology, education, sociology—suffer by reason of a need of the illumination that would follow an extension of psychopathologic methods of research. At present, psychopathology alone studies the integral relation of the individual to his environment. And the attempt to formulate a scientific technic for developing the study of this relation denotes an aim that can command nothing but praise.

But if the aim is praiseworthy, the achievement cannot yet be so described. Directly one turns to psychopathology, one finds that the air is filled with a confusing blizzard of flying words. Unconscious, coconscious, foreconscious, subconscious, islands of consciousness— these are some of the terms that assail the inquirer. When the exact significance of consciousness becomes uncertain, this complicated qualification of consciousness adds nothing to the clearness of discussion. The student has to familiarize himself more or less with the whole area of psychopathologic inquiry before he discovers that the investigation has really brought to light some neglected and extremely interesting facts.

There are at present two psychologies, the academic and the medical, and the methods adopted by the two are radically different. The academician is interested in mental foregrounds; he induces a subject to "think" in order that he may examine the course of his thinking. The medical psychologist is interested in mental hinterlands. If he induces a subject to "think" he does so in order that he may examine other factors in the total situation than the dominant thought. This is the essence of Janet's "method of distraction;" hypnosis, Jung's association test and Freud's analysis of dreams are variations of the same technic. All schools from Janet and Freud to Sidis and Morton Prince have effectively demonstrated the part played in the determination of individual behavior by revery. Academic psychology has tended to the assumption that mental states other than concentrated thinking may be disregarded. Medical psychology has shown that the indivdual organism in every twenty-

four hours alternates between concentration, revery and hypnoid states (including sleep); it has also shown that the real education of the individual, whether normal and intelligent or hysterical and disintegrated, is affected by revery at least as much as by concentration.

In the mood of concentration, the organism is at its highest physiologic and mental tensity, and its relation or response to reality is most direct. (Janet has pointed out that in psychasthenia the chief symptom is inability to achieve either this nervous tensity or the relation to reality.) In the mood of revery or dispersed attention, this tensity relaxes, and the preceding experience is mentally assimilated. When we concentrate, we simultaneously put a preformed response to the test of a new situation and gather material for further revery. When we drop back into revery, we "mark, learn and inwardly digest" what has occurred. This holds true of the scientist in his laboratory, the worker in industry, the child and the savage: there is evidence that it also holds true of such creatures as the horse. There is of course an immense difference between the logical and explicit revery of a mathematician like Poincare and "the deep, undirected meditation of the fisherman and shepherd" (Wallas). But the same generic description applies; the mental life of the savage, the hysteric and the scientist may be stated in terms of the same common denominator. The essential question is as to the relation the individual has achieved between concentration and revery, and by consequence between his responses and reality. Explicit reveries, free from logical irrelevance, mark a nicely discriminated adjustment to the environment: unacknowledged reveries, undiscriminating and irrelevant, characterize the superstition of the savage and the terror of the hysteric. I have adopted the term "primitive idea" to denote the irrelevant and confused meanings in which knowledge and ungraduated response begin. These ideas are characteristic of the infant, the savage, the hysteric and the unlettered man. The "logical idea" characterizes the scientific mind at its best. Such ideas have been disentangled from the mass of irrelevance in which they began; they are characterized finely adjusted responses.

Study of the relation between concentration and revery does much to remove the mystery which at present obscures discussions of memory and amnesia. We habitually and from the first forget events and retain meanings. This applies equally to the scientist and to the hysteric. The Fellows of the Royal Society who figured in Francis Galton's experiment were the leading scientists in England; but they could not remember the arrangement of their respective breakfast tables. Anna O., whose famous case was reported by Breuer and Freud, did not make use of another technic of thinking; she also discarded events and retained meanings. By reflection on experience we reinterpret the world about us from day to day; the objects of our common knowledge take on a behavior significance. We do not need to "remember" meanings, the behavior significance is the object: the world carries its meaning for us, a meaning which is changed only by the development of new meaning (or organic injury or mental disintegration). The difference between the scientist and the hysteric is not that one remembers and the other forgets; it is rather a difference in respect to the nature of the dominating revery and the type of meaning. The reveries of the hysteric are egocentric, emotional and confused; those of the scientist are impersonal, logical and subject to criteria of relevance in thinking. Sometimes it is possible to substitute a healthy for an unhealthy revery theme in the confused reflections of childhood. I know of two instances in which suggestions made during profound sleep by a parent

proved thus efficacious. A girl, aged 3, developed a morbid fear of men in spectacles after being frightened by a motorcyclist in goggles. This persisted for three months, and direct appeal to her childish concentration was fruitless. Three nights of suggestion during sleep banished the fear; during four subsequent years there has been no recurrence. In the other instance, a boy, aged 4, who feared dogs and cats, was similarly aided to readjust his behavior. It is not so much the original experience that determines the individual attitude, it is rather the type of reflection which follows it. It remains true, of course, that the symptomatic fear cannot of itself be banished in the great majority of cases. There is need to explore the primitive idea of which it is the product or manifestation. Improved discrimination, objective criteria and a closer adjustment to externality—the real education lies here. The fears of the hysteric, the superstitions of the savage are self-created; it is the primitive idea, that coagulum of confused experiences, which adds terrors to reality. Where no power of discrimination exists, anything is possible. Incantations and magic alone can reassure.

The "infantile amnesia," discussed by Freud in his "Three Contributions to Sexual Theory" need not therefore delay us. In forgetting the events of his earliest life, the child simply follows the course we all follow until senility or lack of occupation throws our mental life back on itself. In forgetting the meanings he formerly assigned to the world, the child merely illustrates the logical consequences of reinterpretation. As his powers of discrimination improve, his surrounding takes on a new behavior significance; the old significance is not merely forgotten, it becomes unthinkable. In infancy the change is rapid and the repudiation of former beliefs is often surprisingly complete. A small girl, during a period of enforced inactivity and isolation, invented imaginary brothers to bear her company. The characterization, especially of two of them, "Fred" and "Ernest," was vividly real; between the ages of 3 and 5 a great part of her revery thinking was occupied with the brothers' deeds. At the age of 8, when leading a life of normal association with other children, she had completely forgotten the "brothers." I asked her to guess their names and she hazarded "Jack." The readaptation of two and a half years had utterly obliterated "Fred" and "Ernest."

It is therefore the revery after the experience, rather than the experience itself, that determines meaning and individual attitude. The child, like the savage, is unable to distinguish clearly between concentration on externality and revery or dream. (J. G. Frazer in "The Golden Bough" points out that every savage confuses his dream experiences with reality.) Both child and savage consequently confuse, to an extent which we little realize, reality and fiction. Adult standards of reality are of small use to a child. A girl, aged 3, lying in her cot in a darkened room, expressed fear of a black cat. The assurance that no such animal was present gave her no comfort, but when her mother declared that the cat would be chased away the child went to sleep at once. Another child, aged 4, said in a dream "Wouldn't it be nice if the sun shone at night?" The explanation was that shadows became "giants out of fairy stories" in a dim light. It is this confusion of concentration with revery, of actual experience with meditation, that makes the primitive idea.

Primitive ideas are consequently massive structures, compact of irrelevance. The deductions which appear as behavior have, to us, no apparent ground in fact, but are nevertheless grounded in a previous train of revery. Many such ideas persist in the adult, even if he is of average mentality. A married woman,

aged 25, intelligent and mentally well-balanced, suffered a fear of being alone in a house. This resolved itself into a fear of a man breaking in. This fear had a multiplicity of roots in infantile revery. One such was a fear of blindness. At the age of 3, she was constantly told that if she allowed her hair to fall into her eyes she would go blind. At the age of 4, she jeered at the peculiar eyes of another small girl and was told by her mother that such conduct, if persisted in, would lead to blindness in herself. Subsequently, she sustained an injury to the right eye, and some years later was operated on for cataract. Confused with all this was a fear that a blow on the head would cause blindness. A man breaking into a house might silence her with a blow on the head. This indiscriminate confusion of meaning illustrates sufficiently the structure of a primitive idea. In the individual who suffers such an idea, it is not set out clearly and in sequence as I have set it down. The situation is rather that all these things are somehow thought at once in a single complex muddle—that at least is the only way in which I can tentatively describe it. One might say that such an idea reveals itself as a fear and a compulsion which attaches itself to a variety of actually unrelated objects. Any popular superstition provides an illustration. I have seen the part proprietor of a large factory in Philadelphia refuse to leave a business office by another door than that by which he had entered.

Magical ceremonies are the direct outcome of primitive ideas. Such ceremonies are dictated, like scientific research, by a desire for understanding and control. They are, however, based on the irrelevancies of primitive thinking. The savage, says Frazer, fails to distinguish between his shadow and himself, and injury to his shadow consequently arouses the same reaction as an injury to himself. An Anglo-Australian boy, aged 10, who walked a mile to school every day, developed a curious shuffle of three steps, which he invariably performed if anyone chanced to tread on his shadow. Last year in this city I witnessed another illustration of the same primitive idea. An adult seated on a sunny veranda was teasing, not unkindly, a girl of 8. Suddenly the child stamped on the floor. "There," she said, with the conviction of successful retaliation in her tone, "I've jumped on your shadow." Simply one finds convictions that an injury done to a lock of hair will be reflected immediately in the person whose hair it is. Magic is primitive reasoning; the primitive ideas that lie behind such reasoning still go far to determine our civilized destiny. In the *Journal of American Folk-Lore* (January-March, 1923), Miss Beckwith of Vassar recently printed a list of 186 superstitions found in forty-five college girls from literate American families. These superstitions are not all traditional. That every generation makes its own primitive ideas is illustrated by superstitions with respect to pavements, "Step on a crack, break your mother's back," or in examinations, "A seat formerly occupied by a good scholar is lucky." This relating together of irrelevancies is typical of magic and the primitive idea.

The savage is not utterly devoid of ideas that may be termed logical, that is to say, ideas sifted of irrelevancies. Whenever practical crafts (or manipulation) dominate his thinking, his performance is relatively superior. Capt. Pitt-Riveres' photographs of native houses in the island of Awē, off the coast of New Guinea, show that a most primitive people is capable of developing a high degree of technical skill. It remains true, nevertheless, as Malinowski has shown in his account of the Triobrand Islanders ("Argonauts of the Western Pacific") that magic rules not merely every technical operation, but also every

detail of such an operation. New methods of work are consequently acquired with difficulty, not because of any "cake of custom," as Bagehot called it, but rather because any infringement of the rule may be attended by evil magic; in other words, the so-called habits of the savage tend to become "compulsions" under the influence of primitive ideas. This is a fact of special interest to the psychopathologist. Any habit, whether socially imposed or developed by the individual, tends to become a compulsion under the influence of primitive ideas. One sees this illustrated in the child and the Vassar college-girl ("Step on a crack, break your mother's back"); it is probably operative also in the horse which "shies" invariably at a bridge. Even the horse has a capacity for forgetting events and retaining meanings. It is in the mixture of relevant and irrelevant meanings that the primitive idea begins.

Primitive ideas of one sort or another dominate the hidden mental life of the child. Terror dreams and awareness of the wide world beyond the particular experience seem to begin between the ages of 2 and 3. At this age primitive ideas, partly formed long before the child could speak, are actively in operation. Such ideas tend to be apprehensive of possible disaster, because, for the child as for the savage, the wide world which forms the background of every passing experience is unknown and of unexplored immensity. From this time on, fairy stories are especially comforting; the racial lore assures the child that magic words and ceremonies are an adequate precaution against evil powers. Meanwhile, the process of education commences; the civilization of the mind begins in Aristotle's dictum that we must think of one thing at a time, that in every classification there must be a single fundamentum divisionis. When education succeeds, the child is taught to use the essential of scientific method in its thinking; that is, it is taught to state to itself a criterion of relevance and to sift the related from the unrelated, the externally existent from the imagined. How few people succeed in this endeavor is illustrated by the most cursory examination of magazine advertisements. The patent medicine is still directed to the attention of the individual who carries vestiges of primitive ideas. To the ignorant a magic remedy is more potent than a scientific. A known remedy has a limited relevance, an unknown remedy has the boundless possibility of an incantation.

The means by which primitive ideas are logically reconstructed sifted of irrelevance and informed by understanding can be stated to consist in the right use of words. By means of words, the early confused revery is made systematic and orderly. Once things are named, their relation to each other can be thought about in orderly fashion and stated for purposes of critical reconsideration and discussion. Words not only enable persons to communicate meanings to each other, they also enable mankind to amend thought by mutual criticism. Something of this seems to have been in the minds of the ancient Egyptians who identified naming and creation. In their philosophy, Thoth was the distinctively creative manifestation of Tum, the eternal spirit; and Thoth was the authority who named and created things. In speaking of words as thus in a sense creative and as the means to logic, I do not intend to confine my assertion to words in the ordinary sense. Mathematical symbols and chemical formulas are, of course, supreme instances of the thought material of selective logic.

I should like, by way of conclusion, to point out that investigation of the primitive idea and the compulsive action has an importance which extends far beyond the psychopathology clinic. In any individual, be he child, savage, or civilized adult, the struggle to civilize his thinking is greatly affected for good

or ill by the conditions of his immediate surrounding. Set an average person in an adverse environment, and immediately primitive ideas will tend to gain the advantage in his thinking. The only child, or the child of unhappy parents, frequently becomes psychoneurotic in later life. The life of the savage which, so says Hobbes, is "solitary, poor, nasty, brutish and short," is dominated by superstition and magic. In civilization, we tend to neglect the mental effect that long hours and industrial fatigue have on our fellow men employed in factories. After some experience in three continents, I can say that wherever industrial conditions press hardly on humanity I have found primitive ideas at the basis of social and industrial unrest. The primary ill is almost always individual—a demented father in the home, an economic struggle consequent on the death of the bread-winner—but this individual circumstance becomes complicated with the industrial situation in truly primitive and irrelevant fashion. And the solution arrived at is also primitive; there is what a West African would describe as a "smelling out" of witches, a hunt for the "evil spirits" responsible for the ill. The belief that our present civilization is a conspiracy to exploit the working classes is held with paranoid fervor by a large, and possibly increasing, section of humanity. The circumstances which have led modern society to step back toward primitive thinking call urgently for investigation.

THE ORGANIZATION AND DEVELOPMENT OF CLINICAL FACILITIES FOR THE CHILD OF PRESCHOOL AGE. DR. DOUGLAS A. THOM.

Habit clinics, as the term indicates, mean the study and treatment of undesirable habits and personality defects in children, of the preschool age in particular.

The first habit clinic was developed as part of an organization interested in the health of children. The work was carried on for the first year and a half without expense to the Baby Hygiene Association. Last year, with proper funds the work was immediately taken over as an essential part of the medical program of the Association. In the meantime, the Division of Mental Hygiene, which is concerned with the prevention of mental diseases, adopted the habit clinic idea as part of the clinic system throughout the state, and on January 1, a mental clinic for children was placed in a Boston health center, so that, at the present time, clinics for children of the preschool age are part of the medical work of the largest private health agency (Community Health Association) in the State of Massachusetts and the city of Boston.

Psychiatrists have learned that the solution of many of the difficulties of child life is found in a better understanding of the child and that the undesirable habits and personality defects must be interpreted in terms of the child's past experience. It was with this fundamental truth in mind that the development of the habit clinics for children of the preschool age was started.

Appreciating the fact that there is no branch of medicine that lends itself more readily to misinterpretation, it was decided that every habit clinic should be closely associated with some well recognized medical organization and that, in order to eliminate organic conditions as such, a careful physical and neurologic examination should precede admission to the clinic.

The geographical location of the clinics was considered—that is, whether the clinic should operate under one central organization in which all the cases might be assembled for treatment, or whether various units should be established in different sections of the city. It was finally decided that the clinics should be conducted in units and located at such places as to serve conveniently a definite section of the city. This splitting up of the clinic into various units

has developed other advantages, the most important of which has been our ability to cooperate closely with nursery schools, kindergartens, settlement houses and community centers in the immediate vicinity. It is also necessary to have the closest possible contacts with the children's hospitals, child placing agencies, family welfare societies, and such institutions as can give temporary care to children.

Habit clinics must be considered primarily as therapeutic centers and not research laboratories. The success of the clinic depends on therapeutic results, as the physicians, nurses, parents, and social workers who refer most of the cases to the clinics are much more concerned with the beneficial results obtained through the patient's contact with the clinic than they are with any explanation of the underlying mechanisms. It is of interest here to note that frequenly the patients for whom we do most in a therapeutic way are those of whom we have the least understanding from a psychologic point of view. Notwithstanding this confession of inability to understand clearly the mechanisms underlying some of the undesirable habits and personality defects, it is more enlightening to be able to witness the origin and development of these neuropathic symptoms in children than to indulge in the ever-increasing speculative philosophy which is apparently essential when we are confronted with these same difficulties in adult life. In the absence of training, education, and experience, the normal instictive reactions in children lend themselves much more easily to correct interpretations than do the mental conflicts of adult life.

The family, at all times, must be considered as the unit with which we are dealing, for, not infrequently, the individual brought to the clinic is only the product of a defective home. The fault may lie in one or both parents, with an older brother, or a younger sister, or there may be no particular outstanding problem with any of the persons in the home, but the interrelation between family groups may be such as to produce a most unhealthy mental atmosphere.

Not only is it the plan and intention of the clinics to use the simplest methods possible in bringing about the desired therapeutic results, but every effort is made also to formulate such knowledge as we have in simple terms and disseminate it so that it can be utilized by the more intelligent parents, social workers and nurses. For many of the problems, particularly those with reference to feeding, elimination, and sleeping, respond to methods which frequently have been overlooked on account of their simplicity.

It has been interesting to note that many times the symptom which prompts the parent to bring the child to the clinic is by no means the fundamental difficulty in the child's life, but by eliminating this manifest symptom, we are invariably able to hold the parent's interest and have the child continue his visits. For example, in going over the admissions to the habit clinics for a period of one year, I found that only two patients were brought to the clinic whose outstanding problem, in the minds of the parents, was fear; but in studying the cases admitted, fear was found to be the disturbing factor in twenty-four cases.

Any discussion of the child invariably carries with it a long dissertation on heredity, which frequently leaves one in a state of increased perplexity if the author has entertained a nonpartisan point of view, or with some very dogmatic ideas, pro or con, depending on the authority utilized. Kirkpatrick, I believe, expresses the consensus of opinion of physicians dealing with the practical problems of heredity when he states: "From the individual standpoint, heredity should neither be ignored as of no importance nor yielded to as inevitably fixing one's destiny. Instinctive and hereditary tendencies are the roots from which the physical, mental and moral life develops. Some individuals

develop more readily and to a greater degree than others. All are of the same human characteristics, but each may make the most of his environment. Some cannot go as far as others in certain directions nor as easily, but no one has exhausted his possibilities of development. The practical problem is to expend our efforts upon the useful characteristics which we possess in the greatest degree." It seems only reasonable that conservatism permits us to get away from that pessimistic attitude to which the fatalist clings with undying tenacity.

On the other hand, no one, as yet, is ready to say that personality and all its component parts are not molded and colored by social heritage to a great degree and that, after all is said and done, social maladjustments and economic inefficiency are more frequently due to emotional instability than intellectual defects. Therefore, it seems to me that more is to be gained from concentrating our efforts on the study of environment and its effects on the development of personality than can be hoped for by accepting in its entirety the hopeless, fatalistic theory of heredity.

In dealing with the problems presented at the habit clinics success depends on a skill in performance acquired only by study, experience and observation. We must remember, too, that we are lacking in a definite technic in making diagnoses or applying methods such as would be utilized in the treatment of tuberculosis or diabetes.

It taxes the skill and ingenuity of those most efficient to alter many of the home situations with which we are confronted. It is difficult, indeed, to change the moods of parents, their resentful attitudes and indifference, to settle their quarrels and find the underlying cause for depressions, timidity in the mother, arrogance and fault-finding in the father, self-consciousness in a younger sister, and egotism in an older brother. Favoritism by parents is frequently displayed quite unconsciously. One parent may be oversolicitous, while the other may be stern, rigid and unyielding. Common as all these undesirable environmental situations are, they cannot help but play a part in the development of the personality of the child, which cannot be overestimated.

These undesirable habits and personality defects, in order to be eradicated, must be replaced by some new interest. It is not sufficient simply to build up barriers which will thwart the desires and block the emotions acting as the driving force. This force must find a new outlet in a way which is compatible with the environment and satisfying to the child. He should be led and not pushed. All too frequently, it is emotion that acts as the stumbling block rather than the intellect or the will. In order to dispense with undesirable habits and to form new ones, something must be substituted for the rejected habit. The idea of the new habit must be clear in the mind of the child. It must appear to the child as a thing capable of achievement, and the advantages to be gained from its development must be presented in such a way that satisfaction will come with the striving as well as with the accomplishment. The child, handicapped by undesirable habits or asocial tendencies which become more fixed and elaborate and gradually reach out and incapacitate him from meeting the problems of every-day life, becomes a domineering, time-consuming blot on the family life, robbing other children of their just rights. All too frequently, such a child is not only the product but the source of delinquency, poverty and misery in the home. These reactions permeate and contaminate the very foundations of family life.

Many times I have been asked whether I feel that the establishment of clinics for children of preschool age will have any effect on the prevention of mental diseases. My answer to that is simply that, at the present time, we need not justify the work that is being done at these clinics with any vague speculation as to what its beneficial effect may be in ten or fifteen

years. The results that are being obtained from week to week and month to month are of such striking character and of such inestimable value to the children, their parents, and to the community, that we need no other justification for the development of this work as rapidly as personnel and funds can be obtained.

DISCUSSION

DR. EDWARD A. STRECKER: In my paper, I emphasized groups of three: those who were sure to go through life without difficulty, those who were sure to break, and, finally, those about whom nothing can be foretold. I believe "3" has always been regarded as a mystic primitive number. For instance, incantations are invariably repeated three times. After listening to Dr. Mayo's paper, I feel that it is quite important not to leave a primitive number in your minds for which you have no explanation, and which may become submerged in your subconscious minds and, perhaps, later on rise and destroy you. I therefore want to repeat that in speaking of these groups of three, I have a *very definite idea in mind which can be put into words,* thereby robbing it of its mysticism. The group, in which preventive psychiatry should be particularly interested, is the middle group in which, I believe, it is possible to prevent the great incidence of mental disease.

DR. J. HENDRIE LLOYD: The way heredity is treated by some psychiatrists leads to a hopeless position. It practically amounts to fatalism. It is based on the idea of the biologic school which follows Weismann, whose idea is that the germ plasm practically is uninfluenced by the environment; in other words, that acquired characteristics cannot be transmitted. It is a hopeless position, for it means that every living creature is born into a cast iron mold and is going to stay there the rest of its life. Weismann's idea is largely an academic theory in my judgment. There is nothing in this world that is not influenced by its environment. The germ plasm has its environment as much as anything else has. The germ plasm is secreted by a gland of internal secretion. It is nourished, it is subject to the influence of heat, moisture, and above all of nutrition, and the nutritive fluid may or may not contain deleterious or poisonous products; and what is more, it is probably influenced in some obscure way by the so-called hormones, the secretions from the other glands of internal secretion. Now it is perfectly useless to suppose that, with all these environmental factors acting on the germ plasm, it cannot be influenced as well as anything else in nature. Some experiments show that this is true. The male guinea-pig has been saturated with alcohol until he reached a condition of chronic alcoholism, and then allowed to breed. Its young were impaired and epileptic, showing unquestionably the poisoning of the germ plasm in the ancestor. If this can be done by poisons, it seems to me that good can also be done by wholesome influences. I do think, however, that it is necessary in this mental hygiene movement to have an extraordinary amount of optimism. Some of the cases which Dr. Thom referred to require an invincible optimism to make one think that one can do anything with these children. Nevertheless, the work is worth doing. These cases all show the effects of bad environment as well as bad heredity.

DR. THOM: The difficulties which are so obvious in the treatment of the cases that I have cited will not be minimized as time goes on. If we are not able to do anything for these children before they are 6 years of age, it is extremely doubtful whether anything can be accomplished later. Certainly it is during this period that mental activity lends itself more easily to interpretation than it does later in life. The conduct of children is less colored by experience, education and training than is the conduct of adults.

Book Reviews

LA MÉDECINE PSYCHOLOGIQUE. PIERRE JANET, Ernest Flammarion, Paris, 1923.

Professor Janet's latest work is at once a history and a prophecy. It traces the subject of psychotherapy from the miracles at the temple of Esculapius in brief yet graphic style to the beginnings of modern methods, and discusses the present and the future of psychologic medicine. Miracles, animal magnetism, esthesiogeny, hypnosis, psychanalysis, Christian science, new thought, the Emmanuel movement are all submitted to keen analysis. He sketches the neuropathic life of Mrs. Eddy, and then, with characteristic pride in the priority of discovery, points out that the theories of Mrs. Eddy were developed in France by Deleuze and imported into America by Poyen. The same is true of the origin of psychanalysis to which Janet claims fatherhood. Yet he is not especially proud of the traits developed by his child under the hands of Freud, Jung and others; he shows how Freud has altered the terms that he (Janet) employed, has applied the same idea to all cases of psychologic disorder, and finally, "above all has transformed a clinical observation and a therapeutic procedure of precise and limited application into an enormous system of medical philosophy." He does not agree with the theories developed by the freudian school. "Psychanalysis is the latest reincarnation of the magic and psychologic practices that resemble animal magnetism; it has the same characteristics, imagination with lack of critical consideration, over-weening ambition, epidemic spread and a conflict with official science. It is probable that it will also experience unwarranted enthusiasm and decline. Like animal magnetism and hypnotism, however, it will have played a large rôle and given a useful impetus to psychologic studies."

The author takes up seriatum treatment by isolation, reeducation, moralization, the faith cures, work cures, rest cures and others, pointing out strong points here and weak ones there. The historical portion takes up only a minor portion of the book, however, and replete as it is with practical comments, it but leads the way to the modern conception and treatment of the minor mental diseases.

The second part of the book is devoted to consideration of the principles on which psychotherapy is based. However insufficient they may be, all methods have produced some results. The hierarchy of the intellectual processes holds great interest for the author. He states that beneath mature reflection there is a primitive form of feeling that is much more powerful and immediate in its reaction even though less adapted to perfection of the accomplished act. This "immediate assentiment" is the force called on in suggestion and hypnotism; "the essential discovery of the magnetizers and the hypnotists is that by artificial means that produce fatigue and emotion it is possible to bring about experimentally this momentary depression and use it to arouse impulses that are desired. The idea which is introduced into the mind at the favorable moment becomes the object of an 'immediate assentiment' and is transformed into an impulse."

Hypnotization is therefore the bringing about of a lowering of psychologic tension so that tendencies already preformed can become dominant and result in action. The "immediate assentiment" is in obedience to the commands of

the hypnotist, and the subject performs acts which he would not perform were he fully conscious. With this there is dissociation of personal memory, so that not only is there almost complete amnesia for the period of hypnosis, but the tendencies developed during it also disappear after recovery. It is this that constitutes the weak point in treatment by hypnosis and suggestion. During the period of influence the sick man may behave like a healthy one; but later various disorders manifest themselves, and the patient even forgets his period of improved function. "The essence of these treatments is the provocation of automatic acts in place of superior 'reflected' ones. . . I realize that acts executed in this way have real defects. Not being 'reflected' they are not so well adapted to real life, to the situation in hand; they are poorly assimilated in the personality, they leave few memories and do not serve to build up the personality. . . Automatic acts of this kind nevertheless present great advantages for the patient." The patients are delivered from the necessity of thought which is for them so difficult and fatiguing. Even physiologic acts such as eating and defecation are better carried out by these patients when they are relieved from the necessity of thinking about them and perform them under the direction of a physician in an automatic manner. Suggestion plays a similar rôle in the inhibition of purposeless movements, such as tics and spasms.

It is in the section on psychologic economics that the author is at his best. The "budget of the mind" he likens to a financial budget. Certain people are so constituted that they have less mental energy than others. They have a certain amount, ill recognized it is true, of mental energy to use each day, and to overstep the bounds is to run the danger of "mental ruin." He points out that the physician in such cases must ascertain, if he can, not only the income but also the outgo. He must take an inventory of the resources and stop the leaks, find out which actions are exhausting and forbid them, and settle difficulties that are draining the resources of the mental bankrupt.

Acts accompanied by emotion are the most exhausting. The circumstances of life, the continual adaptations necessary in the course from youth to old age, every complication of an action, its rapidity, its duration, its elevation in the psychologic scale, all these are points at which the low-power mind may stall. To this "stalling" Janet devotes an excellent discussion. There are three ways of reattacking a problem that has proved too difficult: going at it again without modification of plan, going at it in a different way and giving it up altogether. Neuropaths seem mentally unable to cope with the problem of finding another way, and yet they are also incapable of resigning themselves to the relinquishment of the problem. They attack it in the same old way, come to the same point, and stall as before. Enormous energy is wasted, and there is none of the satisfaction and increase of mental capital that results from a successful operation. He compares this sequence to a man who invests a certain amount of capital in an enterprise and sees it fail. Then, instead of giving up the idea or attempting to do the same thing in another way, he puts an equal or greater amount of capital into the same concern only to see it fail in the same way. As Janet says, this is positively ruinous.

The great thing in the treatment of neuropaths is simplification of life, "la vie facile." Life must be made routine, ambitions should be renounced, changes eliminated, emotions of all sorts, enthusiasm as well as disappointment, avoided. Life should be a "gray state without emotion." Social existence imposes the most difficult adaptations on the individual. Therefore it is useful and often necessary to remove the patient from his habitual surroundings, his relatives, those who humor him and those who irritate him. Janet remarks

shrewdly that a mania for directing and ordering in one person may go with a mania for service in another who thinks himself above reproach. Thus it is not only the patient who must be treated but the family as well. Above all, neuropaths must be relieved of unsolved problems, of pushing indefinitely against a wall. This is where the liquidation of situations, and especially the liquidation of traumatic memories, comes into play. "After this liquidation . . . the mind ceases to make these efforts at adaptation, which have been repeated indefinitely."

Discipline is also a powerful means of simplifying life. Many neuropaths were healthy during their period of service in the army, and doubtless the convents have saved many minds that otherwise would have gone on the rocks.

In addition to the organization of psychologic economy, the physician in charge of a neurotic person must attempt to increase the income of psychologic energy. This problem presents itself under three heads, "the acquisition of tendencies, the augmentation of the quantity of energy and the elevation of its tension." The acquisition of new tendencies is a tendency in itself. When a problem presents itself, the subject goes about its solution in a haphazard way, expending much energy in useless movements, and by chance strikes on the right combination. The formation of the new tendency consists in conserving useful movements and eliminating the useless ones (the act of swimming might be taken as an example). The acquisition is made easier by a realization of the end to be accomplished, the means available, and by the direction of a person able to instruct. "Since we are very ignorant on the psychology of conduct we teach it very badly." Reeducation is the basis on which new tendencies must be evolved. These patients, however, are also at fault because they have in large measure lost their power of learning; the acquisition of new acts demands great expenditure of energy, and many have not the necessary mental capital even to undertake a useful psychologic investment.

Increase in energy may be brought about in many ways. Certain poisons produce marked outflow of energy, but it is poorly directed. Alcohol, for instance, releases an excessive quantity, but there is a characteristic disorder in its application and at the same time a lowering of tension. Alcohol so often gives rise to increased output of energy that the patients themselves are deceived and say that only when they are drunk do they begin to live like normal people; this is the reason why addiction to alcohol is so common and so difficult to remedy. The succeeding period of depression is an added and especially dangerous factor. There are other remedies of a more psychologic nature. Danger and the absolute necessity of performing certain actions are sometimes curiously elevating and stimulating. A danger passed is like an infectious illness, the patient is immunized. It is probable also that habit spasms and ties originally represented actions that were a source of psychologic income to the patients; but frequent repetition has rendered them useless and even harmful. The mania for domineering has likewise at first a stimulating effect. Even transgressions and crimes may form the basis of exciting remedies; so also may the obsessions for work, exercise, writing and the like.

Of the three principles of excitation, the mobilization of energy in the sense that William James gave it constitutes the first and probably the most important. A major disaster to a business firm demands liquidation of resources to meet the damage. The liquidation is usually excessive and the balance remaining can be used for improvements that will result in larger output and consequent greater income. Psychologic equilibrium constitutes the second principle. A serious illness in many cases, far from bringing complete psychologic ruin, may

make the mental troubles fade into the background and relieve the mind of its previous suffering. The excess energy which goes to combat the illness is ordinarily used up in fretting over the doubts and fears. This excess energy must be turned into good channels that will result in psychologic income instead of unnecessary expense. In addition to the direction of the energy, the tension should be elevated in order to secure psychologic equilibrium. The third principle might be called psychologic irradiation or syntonization. A successful act is followed by delight in its accomplishment and anticipation in forecast for the next. This extra dividend must be utilized before it is frittered away on useless expenditure. The author then shows how these principles of excitation may be employed in psychotherapy.

The third part of the work takes up the application and the results of psychologic treatment. To be effective, treatment must be applied to the proper cases, and for this it is necessary to know something about the disease, the disordered psychology of the patient; in other words, diagnosis must take the first place. Diagnosis does not mean merely classification, for "a classification is never absolute; diseases like living beings form a continuous series which we divide up arbitrarily according to our needs." What is needed is rather a classification according to the portion of the behavior that is disordered, and especially according to the causative factor.

The treatment is more or less complicated according to the nature of the case. Physical remedies of course are employed when necessary, chronic infectious processes being eliminated and so on, but it is more properly with psychologic treatment that the author deals. The man facing bankruptcy sometimes runs the risk of failure because he does not know the sources from which his income is derived and how much remains for future use. In such simple cases an inventory by the physician is often all that is needed to demonstrate to the patient the latent powers he possesses. In cases, however, in which the energies are much reduced the problem is not so simple. Here the treatment consists in rigid economy in expenditure and tension. Rest in bed is good in almost all cases for a short time, especially before the diagnosis is made, and of itself will serve often to clarify the situation. Suggestion, by substituting the "immediate assentiment" for the reflected act and thus lowering the tension under which the act is performed, is often helpful. "The dangers inherent in these treatments have been greatly exaggerated, and I would be disposed to say that hypnotism and suggestion are unfortunately slightly dangerous. I say unfortunately because a remedy is never really powerful unless it is dangerous."

The necessity for caution in the search for memories of traumatisms in the sexual sphere is emphasized, but the author insists on the necessity of ridding the patient of the recurring problem that he is unable to solve satisfactorily. Often, by taking the responsibility on himself, the physician can make the proper decision and bring peace to the patient's mind. Janet warns, however, that in cases of severe asthenia and fatigue from whatever reason, the physician should guard against suggestion, education, psychanalysis and, especially, stimulating forms of treatment. Complete rest, bodily and mental, often for a prolonged period is imperative, and certainly a preliminary to other forms of treatment. The relations between physician and patient are touched on, but the book as a whole is remarkably free from references to the personality of the physician (a sort of magic in itself). Janet is logical in attempting to formulate rules which can be followed by all.

Psychotherapy, however, includes all other methods of treatment; the treatment of a functional nervous case (although the author does not approve of the rigid application of this term) must combine proper physical with the proper mental methods. Drugs are often necessary.

Looking into the future, Janet sees the time when not only the minor mental maladies will be better recognized and treated, but the major psychoses also will be better handled and, especially, prevented. He looks forward to the establishment of mental hygiene clinics for the study of patients who are not in harmony with their surroundings and for the prevention of the development of the minor psychologic illnesses into major psychoses. Mental prophylaxis will take up especially the problem of the "proper administration of the psychologic fortune."

INSANITY AND LAW. A TREATISE ON FORENSIC PSYCHIATRY. By H. DOUGLAS SINGER, M.D., M.R.C.P. (London), Professor of Psychiatry, University of Illinois College of Medicine; Formerly State Alienist and Director of the State Psychopathic Institute of Illinois; and WILLIAM O. KROHN, A.M., M.D., PH.D., Author of "Practical Lessons in Psychology," "First Book in Hygiene," and "Graded Lessons in Physiology and Hygiene"; Formerly Resident Psychologist at Kankakee State Hospital, Head of Department of Psychology at Western Reserve University and at the University of Illinois, and Senior Fellow at Clark University; for six years Medical Juror in Cook County (Chicago) Insanity Court. Pp. 437. Philadelphia, P. Blakiston's Son & Co., 1924.

This book is timely. Nothing on the subject has been published for a number of years, during which time views in regard to the classification of insanity and as to just what constitutes insanity have undergone some modification. The legal tests also have been modified materially in various states and even abroad.

The first part of the book would furnish to a medical student one of the best introductions to the general study of mental diseases. It is written in a clear and simple style, and being furnished with a glossary, is made available to lawyer and layman alike; but the medical point of view is never lost. Forms of insanity are classified along the lines adopted by the American Psychiatric Association. In Part 2, the legal aspects of the subject are taken up in sufficient detail to give the reader a comprehensive view of American court practice, and the variations in different states. Special attention is given to the various crimes in which the insanity defense is likely to be employed, and a useful chapter is furnished on the mental requirements of those making wills.

Altogether the book is well balanced. It furnishes a useful handbook for both lawyer and physician.

ABNORMAL BEHAVIOR. PITFALLS OF OUR MINDS. AN INTRODUCTION TO THE STUDY OF ABNORMAL AND ANTI-SOCIAL BEHAVIOR. By IRVING J. SANDS, M.D., and PHYLLIS BLANCHARD, PH.D. Octavo. Price, $4.00. Pp. 482. New York: Moffat, Yard and Company, 1923.

The title of this book is rather misleading as it deals so largely with psychanalytic phenomena. It is apparently written for the laity, and presents a cursory and unsystematized attempt to cover the entire field of abnormal psychology and neuropsychiatry.

Chapter I on "The Instinctive and Emotional Basis of Behavior" presents a brief elementary review of the psychologic work of E. L. Thorndike, J. B. Watson, and William McDougall, and as a clear and readable resumé, it is one of the bright spots in the volume and has much to recommend it.

In Chapter II, dealing with "Emotional Conflicts in the Causation of Conduct Disorders," case histories are introduced, most of them incomplete and poorly presented. The cases quoted frequently fail adequately to illustrate the points under discussion, and occasionally there is no more than a superficial connection between the two. The same is true in succeeding chapters.

Chapter III, on "Intellectual Capacities and Their Relation to Behavior," deals entirely with the work of others; it is scattered and difficult to evaluate.

In Chapter IV, "The Physical Basis for Behavior and the Influence of Somatic Disease upon Conduct" is presented. The anatomic and physiologic aspects of the human organism, including the endocrins, are taken up. This is done in a sketchy and popular manner, but without making an attempt to substitute simpler language for medical terminology, and the result is probably confusing. Several obvious inaccuracies exist, and a number of debatable points are presented as established facts.

Chapter V, "The Relation of Personality Types to Behavior," presents an approach to the problem of classification of personality types and gives an unbiased, if brief, survey of the conclusions of the pioneers in this work. This chapter is devoid of case histories.

Chapter VI is on "The Rôle of the Psychoses in the Causation of Anti-Social Conduct." Some of the psychoses are discussed in an altogether inadequate and somewhat misleading manner with a variety of quoted cases.

Chapter VII is a companion to the preceding one, but takes up "Borderline Mental Disorders as Causes of Abnormal Conduct." In both of these chapters, one wishes for evidence of greater understanding of the problems involved. The freudian interpretation is given the preference in discussing the psychoneuroses and in the description of the cases; this is true throughout the book.

"Epileptic Manifestations in Behavior Difficulties" receive some attention in Chapter VIII, and the little that is said therein may be accepted without discussion.

Chapter IX, "The Problem of Drug Addition," presents a good resumé of present opinion on this subject, and here one finds the thoughts of previous workers presented.

"The Unbiological Behavior of the Suicide" is the title given Chapter X, and in it are given a number of statistical tables, in part from the work of Fred L. Hoffman on "The Suicide Record of 1917." In general, the presentation is disconnected, and Case 82 on page 357 is not that of a person attempting suicide (self destruction), but an attempt by a dementia praecox patient to escape those whom she believed were her enemies.

In Chapter XI, captioned "Educational Mal-Adjustments," one comes to the keenest realization of the material shortcomings and disadvantages of the book. From the similarity of material and the identical age period involved, one cannot help comparing the cases cited with the masterly and detailed case studies of the Judge Baker Foundation in Boston. One is forced to the conclusion that a single case studied carefully and in detail, as in the latter series, leads to a broader and deeper understanding of behavior difficulties than many volumes such as the present one.

In the twelfth chapter are discussed "Vocational and Industrial Mal-Adjustments." No new lines of thought are evolved, and no mention is made of mass psychology in the production of industrial unrest. One can hardly consider that the surface of this topic is scratched.

The final chapter on "Modern Methods for the Correction and Prevention of Conduct Disorders" consists in part of an historical review of what has been done, and in part, a survey of what is being done by various organizations and agencies in attacking this problem at the present time.

A brief bibliography is appended to each chapter, and in general a good, but limited, selection is made, especially for social service purposes.

General criticism may be made that there is lack of uniformity in the treatment of the subject. The points taken up are not well balanced. At times it is rather complex and heavy with medical terminology or technical terms and in other places more simple. There is a great deal of repetition, and some unrelated facts are mixed with the basic text. The authors have tried to cover too much ground and have overemphasized freudianism. The introduction states that the book is intended as a connecting link between the profession and the laity; it is regretted that the book cannot be considered as filling that gap. The attempt is certainly praiseworthy. There is undoubtedly a sufficient amount of clinical material, as in the case reports, on which to base the substance of a book such as this is intended to be, but the selections are sometimes haphazard and not entirely to the point.